THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—26TH YEAR. SYDNEY, SATURDAY, MARCH 18, 1939. No. 11.

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An Address.[1]

BY THE LATE E. B. M. VANCE,
Sydney.

WE initiate today the first annual general meeting of the infant Australian Orthopædic Association.

From this chair, where by your grace you have placed me, I bid you all welcome, and especially welcome are those who have come from the other States across and half-way across a continent to be here. It is natural enough that Sydney, with its dozen or more orthopædic specialists, should have been the point of origin of this new association. But we all feel that the acorn of today's planting in Sydney will grow slowly (slow growth is generally sure growth) into a sturdy tree, which will in time spread its branches evenly over our whole continent.

In forming an orthopædic association in Australia we have been prompted by the example set by our *confrères* in Britain, America and Europe, where for many years past there have been established national orthopædic associations, which fraternize freely (or as freely as politicians will permit them) and which, were it not for considerations of State, would, I dare say, be bound in a perpetual brotherhood.

"National repugnances do not touch me, nor do I behold with prejudice the French, Italian, Spaniard or Dutch; but where I find their actions in balance with my countrymen's, I honour, love and embrace them in the same degree. I am no plant that will not prosper out of a garden; all places, all airs, make unto me one country", wrote

[1] Delivered at the annual meeting of the Australian Orthopædic Association, March, 1938.

Sir Thomas Browne in his "Religio Medici" three hundred years ago. Most medical men today would subscribe to these words, for we are the most international-minded of men.

One can vividly remember the large and enthusiastic gathering of the British Orthopædic Association, with American and Continental guests, in London in 1924, nor can one easily forget the glowing reports of one's friends who attended a "combined" orthopædic association meeting at Putti's clinic, Bologna, in the same year. One was made to feel that orthopædic surgery was an "eager thing". It is this eagerness, this awareness of the destiny of orthopædic surgery, which has made possible the foundation of this Association in this young country. Thanks are due mainly to the active steps taken by its confident, debonair, younger men.

On looking into the matter we find the debt of gratitude we owe to these older associations, for their pioneering work in establishing orthopædic surgery as a specialty, duly christened and registered amongst the family of specialties to which our mother medicine gives birth from time to time, a maternity which seems enforced and reluctant rather than besought or even acquiescent. In America, in France and in Germany the growth of specialism came before it did in England. Sir Morrell Mackenzie, the laryngologist, writing in the eighties of the struggles of the earlier specialties for acknowledgement in England, said that the reaction to them was that they affronted the dignity of the profession and that the specialist was not considered "respectable". This attitude he explained in an interesting way. In England the professions gained or lost in dignity and repute according as they were or were not connected with the State. The navy and the army wore the King's livery; the church, the law, all wore robes of State on occasions; medicine had none and was anxious to make up for lack of social position by increased "respectability". To whisper that specialism was not respectable was to shoot a poisoned arrow at it indeed.

"In America", still quoting from the same source, "there is no 'leisured class' to look down upon those who labour, the medical faculty is recruited from the best of families in the land, and its members act with an independence unknown in this country. Under these circumstances, specialism, instead of meeting with opposition, was received with open arms."

The American Orthopædic Association has a history of fifty years. When it was founded in 1888 its members were looked upon as mere "strap and buckle" men, that is, mechanicians. X rays, coming about 1900, helped them greatly, and by 1912 they were looked upon as men capable of doing effectual surgery of the limbs, as well as of dealing with mechanical apparatus.

In the early days of the Great War it was Sir Robert Jones, I believe, who sent over a call to the American Orthopædic Association for help, and some twenty-six American orthopædic surgeons responded and gave effective help. After the War the growth of the specialty in America was vigorous, especially in the direction of greater skill in and range of surgical technique. Today the American Orthopædic Association has to limit its membership to the superlatively eminent; in consequence, the American Academy of Orthopædic Surgeons has been formed to absorb the overflow. The last development, in 1935, was the establishment of an American Orthopædic Board consisting of representatives from (*a*) the American Orthopædic Association, (*b*) the American Academy of Orthopædic Surgeons, and (*c*) the Orthopædic Branch of the American Medical Association; and this board conducts examinations for degrees and diplomas in orthopædic surgery.

In England, the British Orthopædic Association began in February, 1918, with Muirhead Little as first President and Sir Robert Jones as Vice-President.

The British Medical Journal, which apparently has never shared the belief which some other English medical journals had, that specialism was not quite the "thing done", published a sub-leader on the formation of the Orthopædic Association, in which it said:

> It is confidently expected that the new body, which has a membership composed of working orthopædic surgeons, and which has determined to require a severe test for the admission of future members, will play a leading part in maintaining the dignity of orthopædic surgery in Britain and in promoting organized scientific and clinical research in this subject.

We all know that Sir Robert Jones, who was for the six succeeding years President, played a vital and vigorous part in establishing the British Orthopædic Association and in promoting harmonious relations with other national associations. We feel that his spirit is with us today in our efforts to establish the Australian Orthopædic Association.

In the words of our constitution the aims of our association are:

> (1) The purpose of this Association shall be the advancement of orthopædic surgery. (2) To provide a national and authoritative body of experts to act, advise and cooperate in all matters affecting the welfare of cripples, and the prevention and relief of crippling disabilities arising during peace or as the result of war.

And the condition of full active membership is set out as follows:

> Active membership shall be limited to those practising orthopædic surgery exclusively, and any such member shall be a member of the British Medical Association or shall be eligible for membership of that association.

It had been found by previous inquiries that only by making this a condition of active membership could the association be admitted to the privileges of affiliation with the other national associations. Over Plato's academy was inscribed: "Let no one who is not a geometrician enter." Over our academy, with the substitution of the word "orthopædician", we have in effect the same inscription.

Why specialize? Why associate? Why nationalize the associations? These are questions which arise in the mind.

Why do we specialize? Of medical science as applied to its subject, the human body, Osler has said: "Its problems are more complicated and more difficult than any others with which the trained mind has to grapple." As a machine of ten thousand named anatomical parts, it would be complex enough, even if the maker were human and could be called to account. But as this is impossible, and the physiological laws of its functioning are beyond our knowledge in full, then the permutations of derangement are inexpressibly numerous. A large amount of our work remains in the realm of conjecture because we cannot measure up all the factors in it. Add to this that medical science is progressive and a continuous stream of new facts is being poured into it.

We are thus confronted with a field of boundless extent for the object of our study.

"It is impossible for a man who attempts many things to do them all well" is a truism; but it was first said by Xenophon a long while ago, and has therefore stood the test of time.

The largeness of the field and the limitations of the human intellect complete the argument.

This state of affairs is common knowledge and leads logically to subdivisions of a field so vast. The inevitability of this is so well recognized nowadays by the profession, members of which may or may not regret it, and by the public, who demand it, that it is unnecessary to pursue the matter further. The question of today is not so much "why specialize?" as "where specialization is to end".

Apropos of advanced specialism, Herodotus says of the ancient Egyptians: "So wisely was medicine managed by them that no doctor was permitted to practise any but his own branch; some were oculists, others attended solely to complaints of the head, others to the teeth, and some to the secret maladies of the abdomen." So the age of specialism is not new.

One aspect of specialism which has struck me is that the presence of specialists (and better still, an association of specialists) in a State tends to improve the standard of general knowledge of the general practitioners in that State. In this association, for instance, we shall concentrate on the smaller subject, but shall gain more knowledge and skill in it. This we are in duty bound to pass on. What we learn must be published and taught, and thus through accredited channels reach the profession, thereby raising the standard of knowledge. Through members of the medical profession knowledge will reach the public, who are interested in knowing what can be done to prevent or correct a deformity.

As to why we should form associations, one can say that the main reason for any medical society is the need for continuing our medical education. This is necessary for us all to the end of our days. To listen to a prepared paper is good education; the preparation of it is even better education. The formation of subcommittees to collect evidence on the end results of certain lines of treatment is good education. So too is the demonstration of cases at clinical evenings.

We form associations, too, to make a joint stock of our knowledge; thus each of us improves his knowledge and consequently his position. "For the strength of the pack is the wolf, and the strength of the wolf is the pack." This, says Kipling, is the law of the jungle, and it is with orthopædicians as it is with wolves.

In politics, medical or State, our association becomes a party, therefore, to be reckoned with—able to declare policies and to bargain if necessary.

We feel the imminence of moves to nationalize the medical profession. In these moves we must be prepared to take a part. Then, too, there is the association's function of being an agency for our scientific, clinical and teaching efforts. By Hippocratic oath and by tradition we are enjoined to hand on the torch to younger men. If we have, as we know we have, laid hold upon some portions of truth in our domain, we must provide proper facilities for passing this on to those who follow. Both undergraduate and post-graduate teaching should receive our attention fairly soon.

I hope that this association will stand for community of ideals and that it will act as a social meeting ground—the place where the realities of keen competition and, shall we say, brotherly jealousies can be dispelled in the genial warmth of friendship.

And finally, amongst the assets of the association is this matter of affiliation with kindred societies abroad. Affiliation leads to an interchange of journals, reciprocal courtesies and personal communication, useful both in our lives and work here and very useful in our travels abroad. Situated as we are in Australia, remote and perhaps a little unfriended, travel as a means of education in our specialty is so highly desirable as to be almost imperative. For a surgeon going with letters of introduction as a member of the Australian Orthopædic Association would, we know, secure a degree of consideration and lead to an exposition of work which would not be granted with the same gusto to the unlabelled peripatetic medical man. When one says this, one feels a little face reddening when one remembers the warmth of greeting as a rule extended to medical men by medical men anywhere, any time.

This association will lead to, we hope, official international visits. It has already been a great pleasure for us to welcome Dr. Kellogg Speed from Chicago and to have him address us on a new operation for spondylolisthesis. What a stimulus it would be to have a person like T. P. McMurray or a Naughton-Dunn pay us a visit.

If we do not have these interchanges we shall be in danger of becoming "the same persons, telling

to the same people the same things about the same things", like imprisoned men, for the rest of our lives.

Native wit, a flair for healing, good teaching, private thought, personal reading, friendly chatting with colleagues, each plays a part in building the better doctor each of us hopes ever to be. But there would still be one thing missing if we did not have the inspiration of a sense of personal contact with fellow workers and masters of our craft in other lands. The gap cannot be filled with reading the writings of these men. For sometimes the better writer may be the poorer man of action, and in surgery it is action which speaks louder than words.

In the matter of naming our orthopædic associations with a national adjective, American, French, Czechoslovakian, Australian and what not, there is more than meets the eye. These are not merely convenient place names. They convey to the discerning that, in the growth of orthopædic art, which is international, contributions come from the several nations, and that each contribution has an intrinsic quality—a smell of the soil from which it sprang.

Is there not in Stromeyer's tenotomy and Vulpius's muscle transplant a certain German ruthlessness; in Putti's arthroplasty of the knee and in the cineplastic operations of the Italians a manual dexterity which well accords with their proficiency in sculpture. In the use and application of plaster of Paris one sees a French artistry. In America there is a capacity for taking no end of trouble and expense in diagnosis and treatment, with a flair for the mechanical side of orthopædics. In Lister and Thomas one sees the British genius exemplified. What is it? Perhaps a capacity to penetrate deeply into the natural processes and to establish underlying principles.

To travel and to absorb these essences and to blend them in one's practice is a worthy ideal. In cuisine, in wine, in whisky, in tea, in perfumery, the best effects are produced by blending. Why not in orthopædics?

What can be said for the chances of a natural growth of Australian orthopædic art? There are men in this room whose contributions are important, though perhaps not peculiarly Australian. But we have in Colin MacKenzie's work, and set out in his book "The Action of Muscles", a splendid starting point for a native orthopædic art. This native art will be characterized by a devotion to the problems of muscles and movement rather than to bones and form. This latter has rather been the point of view in the past, as the title *Journal of Bone and Joint Surgery* bears witness. When our own journal comes I hope it will be called *Muscle and Motion Surgery*.

The study of comparative anatomy, especially that of the Australian fauna, is shown by MacKenzie to be a fountain of useful ideas in the practical problems of surgery. It is indeed a good omen that we have in our list of papers for this meeting one entitled "Comparative Anatomy of the Knee Joint".

May there not be a possibility, too, of regionally characteristic contributions from Australian States—something solid and sombre from Melbourne; some far-reaching horizon-wide generalization from Western Australia; some cultured criticism from Adelaide; and as for Queensland, that vast land which is to us what Africa was to the ancients, it is a country from which something unexpected might come at any time.

After this flippancy I humbly beg your leave, gentlemen, to finish my address to you foundation members of the Australian Orthopædic Association with a dignified quotation. It is from Saint Paul, writing to one of his societies of early Christians. He abjures them, as I do you:

> Let us hold fast the profession of our faith without wavering; and let us consider one another to provoke unto love and good works; not forsaking the assembling of ourselves together, but exhorting one another; and so much the more as ye see the day approaching.

THE CONTROL OF THE DISORDERED METABOLISM IN HYPERTROPHIC PYLORIC STENOSIS OF INFANCY.[1]

By HOWARD WILLIAMS, M.D.,
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THIS paper is a preliminary report of the investigation of the problems of disordered metabolism occurring in infants affected with congenital hypertrophic stenosis of the pylorus. Methods of controlling this disordered metabolism are discussed and a new method is suggested.

A clinical and biochemical study of infants with pyloric stenosis reveals that many are in a state of poor nutrition and in an abnormal biochemical condition. A disordered chloride metabolism and a state of alkalosis and dehydration have been demonstrated in such infants by a number of workers. The depressed nutrition of many of these infants is attributable to loss of food by vomiting; but defective maternal nutrition during pregnancy may be a factor of importance. It is emphasized that these changes vary considerably in individual cases of pyloric stenosis.

The standard treatment of pyloric stenosis in infants at the Children's Hospital, Melbourne, is an operative relief of the obstruction within a few days of the establishment of the diagnosis. Amelioration of the abnormal biochemical state should be undertaken in each case as a preoperative measure.

The changes mentioned above will be discussed more closely, and the extent to which they occur and are amenable to correction will be indicated below.

The under-nutrition due to food loss by vomiting is most rapidly relieved by an operative procedure. In certain infants whose blood protein content is

[1] Read at a meeting of the Melbourne Pædiatric Society on October 12, 1938.

deficient, and in those suffering from anæmia, improvement can be effected by transfusion of blood serum or whole blood. The question of blood transfusion is at present receiving special attention, with respect particularly to the indications for this measure and the value attaching to it.

The chief clinical manifestations of dehydration are the wizened, wrinkled, inelastic condition of the skin, poor tissue turgor, and sunken eyes and fontanelle, although actual loss of subcutaneous tissue may also be a factor in the determination of the external appearance of the body. The high hæmoglobin percentage, the high erythrocyte count and the high hæmatocrit reading provide evidence of hæmoconcentration due to loss of fluid from the tissues and plasma. In general, the degree of dehydration runs parallel with the severity and the duration of the vomiting. This state of dehydration can be corrected very well by the suitable administration of normal saline solution, as will be demonstrated below.

Several biochemical aspects indicative of disordered chloride metabolism have been investigated, notably by Graham and Morris.[1] They showed that the plasma chloride content is lowered, and that there is almost a complete absence of urinary chloride, and they suggested an association between the two observations. They also estimated the chloride content of the various tissues of infants who had died from pyloric stenosis and to whom no saline solution had been administered therapeutically. In all their cases the chloride content of the tissues was very much reduced, in some to a figure below 50% of normal. The view that a universal chloride deficiency existed was further supported by the observation of Graham and Morris that injections of approximately one or two grammes of sodium chloride intravenously or subcutaneously resulted in almost complete retention of chloride, whereas in normal infants proportions varying from 50% to 90% were excreted in the urine.

Gamble and Ross have demonstrated that a similar disordered state of chloride metabolism occurs in experimental obstruction of the pylorus in dogs. Many other workers have made similar observations in connexion with obstructive lesions in the region of the pylorus in man.[5]

Closely associated with the deranged chloride metabolism is a non-gaseous alkalosis with an increase in the carbon dioxide content of the plasma. Graham and Morris[2] have shown that the alkalosis in pyloric stenosis is closely related to and runs parallel with depression of respiration, which may be manifested clinically by slow, shallow respirations, periodic breathing or apnœic periods, or by a combination of them. The rather drowsy, lethargic state of many of these infants is also probably related to the alkalosis. Graham and Morris[3] have admirably represented schematically the alteration of the acid-base metabolism in pyloric stenosis, and a slightly modified form of their graph is reproduced in Figure I.

Gamble has shown in his experiments on animals that the alkalosis and altered chloride metabolism may reasonably be attributed to the loss of chloride in the vomitus. The increase in carbon dioxide content appears to be an important part of the body's method of maintaining the ionic concentration of the blood at normal level and simultaneously compensating the deficiency in the acid radicle. The facts that chloride is lost in the vomitus and that this derangement of metabolism can be corrected by the administration of saline solution, are fairly strong points in evidence that the theory is correct.

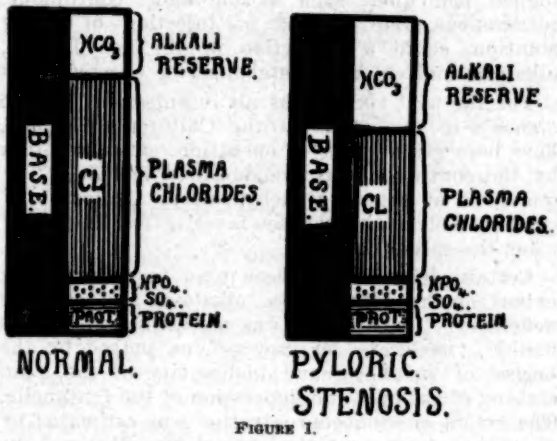

Figure I.

In pyloric stenosis the levels of concentration of the bicarbonate and chloride ions are usually in inverse relationship; but this is not invariably the case. Graham and Morris[1] believe that loss of chloride in the vomitus in pyloric stenosis is an important factor in the production of the alkalosis and altered chloride metabolism, but that that is not the only one. These workers have shown that a restoration of the blood chloride content to a normal figure by injection of saline solution intravenously or subcutaneously does not result in a correction of the alkalosis; this is proved by the persistence of the high total carbon dioxide content of the blood and depression of breathing. They have shown also that after an intravenous injection of approximately one gramme of sodium chloride, although the chloride level of the blood returns to normal, as little as 5% is excreted in the urine. However, there is reason to believe that chloride loss in the vomitus is the prime factor, for it has been shown that in the majority of cases a return of the disordered metabolism to normal can be brought about by the administration of normal sodium chloride solution, by the continuous intravenous slow drip method, larger quantities than Graham and Morris used being given.

Methods of Preoperative Treatment in Pyloric Stenosis in Infants.

The methods hitherto adopted of attempting to correct the altered metabolism and to improve the

preoperative condition of these infants have been the subcutaneous, intraperitoneal or intravenous injections of normal saline solution, in amounts ranging from 100 to 150 cubic centimetres, and at times the parenteral injections were repeated for the same patient. It has been found that such small injections have no significant effect on the alkalosis, but may give rise to a temporary elevation in the plasma chloride content.[1]

Consideration of the extent of tissue depletion of chloride, and the fact that loss of chlorides and of water had been in progress for some time, suggested to us that only by a mechanism based on physiological principles, such as the slow, continuous, intravenous drip method of injection of saline solution, could a correction of the dehydration, alkalosis and chloride metabolism be effected.

For the past six months all infants with pyloric stenosis in the wards of the Children's Hospital have been given, prior to operation, saline solution by the continuous intravenous drip method. The questions of degree of dehydration, alkalosis and chloride deficiency have been investigated before and after the saline therapy.

Certain criteria have been used to estimate the extent of the dehydration, alkalosis and chloride deficiency. Dehydration was assessed clinically as "mild", "moderate" or "severe", as judged by the degree of wrinkling and inelasticity of the skin, sinking of the eyes and depression of the fontanelle. The extent of hæmoconcentration was estimated by the erythrocyte count, the hæmoglobin value and the hæmatocrit reading. Alkalosis was estimated by the carbon dioxide combining power of the plasma, and clinically by the degree of depression of respiration, the degree of lethargy and of drowsiness. Chloride deficiency was measured by the level of plasma chlorides and by the proportion of chloride in the urine.

The results in cases investigated are recorded in Tables I and II. In Table I are shown the alkali reserve and plasma chloride content immediately before the institution of the continuous administration of saline solution, and again immediately afterwards, the amount administered and the duration of administration being indicated. These values were estimated again just prior to the infant's discharge from hospital, and are tabulated in the columns on the right.

In Table II are shown the hæmoglobin value, red blood cell count and hæmatocrit reading before and after saline therapy, and again at the conclusion of treatment in hospital.

These estimations and their clinical significance are discussed later; but it will be noted that the figures after saline therapy correspond fairly closely in most instances to those registered when the infant was discharged from hospital. This suggests that physiological normality was attained before any surgical measures were undertaken.

The Continuous Intravenous Administration of Saline Solution.

The continuous intravenous administration of saline solution is not difficult to carry out, provided standard equipment, such as that available in all the wards at the Children's Hospital, Melbourne, is used. A short metal needle with stilet for inserting a cannula into the vein is a most important factor. This needle is the same as that described by Collins[7] in the issue of The Medical Journal of Australia of December 31, 1938, in connexion with transfusion of fluids to infants. It is our usual procedure to employ one of the antecubital veins, for we have found that the saline solution runs more easily in one of these veins than in the internal malleolar vein at the ankle. Furthermore, the arm is more easily splinted than the leg; but the veins are not so readily found as is the internal malleolar vein. It may be emphasized that with the apparatus described and with careful technique, little or no difficulty has been experienced in the administration of the saline solution to small

Table I.

Showing Alkali Reserve, Expressed as Volumes of Carbon Dioxide per 100 Cubic Centimetres of Plasma, and Plasma Chloride Content, Expressed as Milligrammes of Sodium Chloride per 100 Cubic Centimetres of Plasma.

Initials of Infant.	Age in Weeks.	On Admission to Hospital.		After Administration of Normal Saline Solution.				On Discharge from Hospital.		
		Volumes of CO_2 per 100 Cubic Centimetres of Plasma.	Milligrammes of Sodium Chloride per 100 Cubic Centimetres of Plasma.	Amount of Normal Saline Solution (Cubic Centimetres).	Hours Taken for its Administration.	Volumes of CO_2 per 100 Cubic Centimetres of Plasma.	Milligrammes of Sodium Chloride per 100 Cubic Centimetres of Plasma.	Number of Days between Operation and Discharge from Hospital.	Volumes of CO_2 per 100 Cubic Centimetres of Plasma.	Milligramme of Sodium Chloride per 100 Cubic Centimetres of Plasma.
E.T.	6	70	542	600	24	56	590	18	59	622
L.P.	6	74	490	900	36	65	578	14	61	603
R.M.	3	94	424	1470	48	65	600	12	61	619
P.L.	3·5	72	518	1200	48	57	618	59	42	620
D.F.	4	88	440	1260	28	65	600	10	58	582
J.M.	5	96	402	1260	40	63	550	12	58	612
J.C.	3	86	480	810	15	64	592	19	64	540
W.Mc.	8	90	422	1350	48	72	562	14	64	598
B.T.	6	78	498	1470	48	60	572	Death 2 weeks after operation.		General peritonitis.
J.D.	6	126	424	750	24	80	558	11	60	590
A.J.	4	158	558	960	40	76	564	17	64	612
E.F.	3·5	90	450	1260	48	76	560	42	44	600
G.H.	7	80	578	600	24	50	604	24	55	580
P.A.	7	79	480	600	23	63	578	18	53	602
K.S.	4	68	546	450	15	57	626	12	59	600
D.T.	4	93	476	1050	40	64	588	20	52	594

TABLE II.[1]

Hæmoglobin Value, Erythrocyte Count and Hæmatocrit Reading.

Initials of Infant.	Age in Weeks	Before Saline Therapy.			After Saline Therapy.					On Discharge from Hospital.			
		Hæmoglobin Percentage.[2]	Erythrocytes. (Millions per Cubic Millimetre).	Hæmatocrit Reading.[3]	Amount of Normal Saline Solution. (Cubic Centimetres).	Hours Taken in Administration.	Hæmoglobin Percentage.	Erythrocytes. (Millions per Cubic Millimetre).	Hæmatocrit Reading.	Number of Days Between Operation and Discharge from Hospital.	Hæmoglobin Percentage.	Erythrocytes per Cubic Millimetre.	Hæmatocrit Reading.
E.T.	6	100	—	40	600	24	75	—	32	18	64	—	28
I.P.	8	84	—	37	900	36	68	—	31	14	66	—	30
E.M.	3	118	—	47	1470	48	83	—	39	12	74	—	36
P.L.	3·5	122	—	49	1200	48	90	—	41	59	78	—	34
D.F.	4	112	—	46	1260	28	82	—	36	10	72	—	32
J.M.	5	100	—	42	1260	40	84	—	36	12	66	—	28
J.C.	3	114	5·9	44	810	15	90	5·0	40	19	75	4·6	33
W.Mc.	8	100	6·0	42	1350	48	77	4·75	34	14	65	4·1	29
B.T.	6	118	5·9	46	1470	48	93	5·2	40	Death two weeks after operation. General Perito nitis.			
J.D.	6	100	5·5	39	750	24	80	4·6	34	11	78	5·0	35
A.J.	4	128	6·1	49	960	40	92	4·8	35	17	77	4·6	32
E.F.	3·5	130	6·7	52	1260	48	112	5·7	47	42	65	3·6	28
G.H.	7	80	4·5	35	600	24	72	4·5	32	24	65	3·8	28
P.A.	7	90	5·0	36	600	22	68	4·4	27	18	64	4·5	27
K.S.	4	125	6·9	50	450	15	105	5·6	45	12	94	4·9	40
D.T.	4	110	5·9	45	1050	40	75	4·6	34	20	62	4·0	28

[1] The children referred to are the same as in Table I and in the same order.
[2] The hæmoglobin value is expressed as a percentage (13·8 grammes=100%).
[3] The hæmatocrit reading is expressed as an absolute value of packed erythrocytes per 100 cubic centimetres of blood.

dehydrated infants. The flow was regulated so that the child received from 15 to 30 cubic centimetres (half an ounce to one ounce) of fluid per hour, according to the size of the infant, the extent of dehydration and the general nutrition. The more sick the child, the slower should be the rate of administration of the saline solution.

The amount to be administered is best estimated by a careful examination of the child from time to time. When the clinical condition has improved, as judged by the disappearance of wrinkling, by a return of the elasticity of the skin to a more normal tissue turgor, and by a return of the respiration rate and of the drowsy state to normal, then the saline solution may be discontinued. Amounts varying from 750 to 1,500 cubic centimetres (25 to 50 ounces) have been considered necessary, according to the clinical condition of the infants. These amounts appear safe to administer, and the only ill-effect noted has been the occurrence of mild œdema in two cases. This was possibly due to excessively rapid administration in the early stages, for each child received from 150 to 180 cubic centimetres (five to six ounces) of saline solution in fifteen minutes. It is very important that the saline administration should be suspended immediately on the appearance of any œdema.

Discussion.

This investigation has been concerned primarily with a study of the means of making better "operative risks" of infants suffering from pyloric stenosis.

The estimation[1] of the extent of the alkalosis (expressed in volumes of carbon dioxide per 100 cubic centimetres of plasma) and of the chloride deficiency (expressed in terms of milligrammes of sodium chloride per 100 cubic centimetres of plasma) agree reasonably well with the figures of other workers. In all cases the findings have been abnormal. The common findings were: an alkali reserve of 85 to 100 volumes per 100 cubic centimetres of plasma (normal values, 50 to 60 volumes per 100 cubic centimetres of plasma), and a plasma chloride content of 400 to 450 milligrammes per 100 cubic centimetres of plasma (normal values, 560 to 620 milligrammes per 100 cubic centimetres of plasma). In all but a few instances the alkali reserve was increased in proportion to the decrease in the plasma chloride content. This suggests that the bicarbonate ion replaces the chloride ion, and by so doing maintains the ionic balance in the blood and neutralizes the base liberated by the loss of the chloride ion.

Case XI is an example of an alkali reserve being disproportionately high in comparison with the plasma chloride content.

A.J., aged four weeks, was found to have an alkali reserve of 158 volumes of carbon dioxide per 100 cubic centimetres of plasma and a respiration rate of 10 per minute; but the plasma chloride content was not proportionately lowered, being 558 milligrammes per 100 cubic centimetres of plasma. Administration of 960 cubic centimetres (32 ounces) of normal saline solution over a period of forty hours reduced the alkali reserve to 76 volumes of carbon dioxide per 100 cubic centimetres of plasma, and the plasma chloride content rose to 564 milligrammes. With these biochemical changes there were a corresponding clinical improvement and a return of the respiration rate to 26 per minute.

The reason for this very high alkali reserve without a corresponding fall in chloride content is very difficult to determine, and as yet no satisfactory explanation has been advanced.

In the majority of cases the degree of dehydration, as estimated clinically, and by the hæmoglobin,

[1] The analytical methods used were the following: the alkali reserve was calculated by the Van Slyke method, and the plasma and urinary chloride contents were calculated by the Whitehorn methods; the hæmatocrit readings were made according to the method described by Wintrobe;[10] the hæmoglobin value was estimated on the standard Sahli apparatus, and the erythrocyte count was made with the Thoma-Zeiss counting chamber.

hæmatocrit and erythrocyte readings, corresponded with the degree of alkalosis and chloride deficiency. A moderately accurate estimation of these changes can be formed on clinical grounds if one looks carefully for the signs of alkalosis and dehydration, to which reference has been made earlier. Biochemical estimations are not necessary in most cases; they form a valuable guide, but a guide only, in the assessment of the clinical picture.

After clinical appraisement of these cases, and correlation with the biochemical results, it is realized that in a number of instances the degree of alkalosis, chloride deficiency and dehydration does not explain the whole clinical picture. As previously mentioned, the factor of under-nutrition plays an important part. It is readily understandable that the control of this factor is extremely difficult. Better nutrition of the pregnant or nursing mother, earlier diagnosis, and possibly transfusion of blood to replace deficient protein are measures that may be of considerable help. However, we are concerned here primarily with the preoperative control of the dehydration, alkalosis and chloride deficiency.

Three facts are emphasized: (i) that the return to normal of the disordered metabolism after operation is slow in patients who have had no preoperative therapy; (ii) that a small injection of saline solution produces no clinical or biochemical change of real significance; (iii) that administration of large quantities of saline solution by the continuous intravenous drip method produces very decided improvement both clinically and biochemically. These points will be discussed in some detail.

(i) It has been possible to show in cases of pyloric stenosis, in which no saline solution has been administered preoperatively, that a period of approximately eight to sixteen days elapses after operation before the chloride metabolism, alkalosis and dehydrated state return to normal. Three cases have been investigated, and the biochemical changes have been estimated at suitable intervals of time after operation. The following is an example:

C.A., a male infant, aged four weeks, of healthy parentage, was admitted to hospital with a history of projectile vomiting and constipation of nine days' duration. He was a drowsy infant, in a fair state of general nutrition, weighing 3·6 kilograms (seven pounds thirteen ounces). The respirations numbered 18 per minute. Gastric peristalsis was visible through the abdominal wall, and a pyloric tumour was palpated. No saline solution was administered preoperatively. A modified Rammstedt's operation was performed under local anæsthesia, a large tumour being found. The post-operative course was uneventful, the infant vomiting on only one occasion. In Table III are shown the alkali reserve, plasma chloride content, urinary chloride content, hæmoglobin value, erythrocyte count and the hæmatocrit reading, estimated before operation, and after operation on the second, seventh and fourteenth days.

The clinical picture of dehydration and alkalosis corresponded reasonably well with the biochemical findings. The gradual return to normal over a period of fourteen days is demonstrated from the figures. This slow return to normality was not due to vomiting, for the infant vomited on only one occasion after operation, and the post-operative course, apart from a very mild degree of wound sepsis, was uneventful. Coincidentally with the biochemical improvement the clinical condition improved, and the infant was discharged on the fourteenth day.

(ii) It has been shown that the disordered metabolism is not restored to normal by the administration of small amounts of saline solution. Graham and Morris[1] have shown that, after the intravenous injection of approximately one gramme of sodium chloride in solution, a temporary elevation of plasma chloride content usually occurs, but that no change in the alkalosis results.

A study of three of our cases in which only small amounts of saline solution were used, showed similar results to those of Graham and Morris. No significant clinical improvement was noted in these cases after following the administration of small amounts of saline solution. The details of such a case are as follows:

S.R., a male infant, aged ten weeks, of healthy parentage, progressed favourably for seven weeks, and then began

TABLE III.

Showing Slow Return to Normal Values of the Alkali Reserve, Plasma Chloride Content, Urinary Chloride Content, Hæmoglobin Value, Erythrocyte Count and Hæmatocrit Reading, in the case of an Infant, C.A., aged Four Weeks, treated Surgically for Pyloric Stenosis, but having Received no Saline Therapy.

	Alkali Reserve: Volumes of CO_2 per 100 Cubic Centimetres of Plasma.	Plasma Chloride Content: Milligrammes of NaCl per 100 Cubic Centimetres of Plasma.	Urinary Chloride Content: Percentage.	Hæmoglobin Value Percentage. (13·8 grammes = 100%.)	Erythrocytes: Millions per Cubic Millimetre.	Hæmatocrit Reading.
17/3/38	106	526	Nil	115	6·2	48
20/3/38			Modified Rammstedt Operation.			
22/3/38	102	472	Nil	90	5·3	39
27/3/38	88	558	0·008	85	4·7	37
3/4/38	70	608	0·008	78	4·5	34

vomiting in a forcible manner after almost every feeding. When examined at hospital he was drowsy, and his respiratory rate was 17 per minute. Unmistakable gastric peristalsis was observed, and a pyloric tumour was palpated. He presented clinical evidence of mild dehydration only. After the administration of 180 cubic centimetres (six ounces) of normal saline solution by the intrafontanelle route no appreciable change either clinically or biochemically was noted. The alkali reserve, the plasma and urinary chloride content, the hæmoglobin value, the hæmatocrit reading and the erythrocyte count are tabulated in Table IV.

(iii) In contrast to the absence of therapeutic results obtained by the use of small quantities of saline solution, the intravenous administration of large amounts of normal saline solution by the slow drip method has resulted in a return to approximately normal values of the alkali reserve and the plasma chloride content. The dehydration was relieved in every case so far investigated. These biochemical changes are tabulated in Tables I and II, and the figures obtained on the discharge of the infant from hospital may be used for purposes of comparison. Coincidentally with these biochemical modifications a striking clinical improvement occurred. This was indicated by a filling-out of the subcutaneous tissues, a return to normal of the respiratory rate, and an improvement in the drowsy, lethargic state of the infant.

From these cases it can be demonstrated that the alkalosis may be corrected by saline therapy. This is contrary to the findings of Graham and Morris, and suggests that Gamble's view of alkalosis arising from chloride loss is the correct one. The results indicate that chloride loss is the factor of prime significance, and that by the administration of sodium chloride the disordered metabolism can be restored to normal in the majority of cases. It seems reasonable to assume that only by the slow, continuous and prolonged administration of sodium chloride can an ionic interchange between tissue cells and tissue fluid and blood be made to occur and thus correct the state of alkalosis and chloride deficiency. The continuous intravenous drip method is one which attempts, with considerable success, to fulfil this physiological principle of gradual change.

The continuous intravenous administration of saline solution to an infant calls for a warning. The rate must be slow; between 15 and 30 cubic centimetres (half an ounce and one ounce) should be given per hour. A careful watch must be kept for any sign of œdema, particularly bagginess under the eyes or œdema of the scrotum; and on the appearance even of slight œdema the administration of the saline solution must be interrupted immediately. Evidence of œdema occurred in only two out of eighteen cases, and in both of these between 150 and 180 cubic centimetres (five and six ounces) of saline solution were inadvertently administered within a period of fifteen minutes.

A number of these infants when fully rehydrated have a certain degree of anæmia. This usually responds very well, in the course of a few weeks, to the administration daily of 0·1 to 0·2 gramme (one and a half to three grains) of ferrous sulphate. In a few cases, specially selected on account of poor general nutrition and anæmia of the infant, a blood transfusion has been a valuable therapeutic measure.

Conclusions.

1. Infants with pyloric stenosis exhibit varying degrees of dehydration, alkalosis, disordered chloride metabolism and under-nutrition. The extent of these changes varies considerably in individual cases, and the different changes are not necessarily of a similar degree.
2. In the assessment of the severity of these changes clinical appraisement of the infant is very important in addition to the biochemical investigation.
3. The dehydration, alkalosis and disordered chloride metabolism of patients treated surgically, but without the pre-operative administration of saline solution, were alleviated slowly.
4. As a means of combating the alkalosis, dehydration and disordered chloride metabolism, single small injections of saline solution are of little value as judged clinically and biochemically.
5. The administration of saline solution by the continuous intravenous drip method is a valuable

TABLE IV.

Showing the Absence of any Significant Change in the Alkali Reserve, Plasma Chloride Content, Urinary Chloride Content, Hæmoglobin Value, Erythrocyte Count and Hæmatocrit Reading, in the case of an Infant, S.R., aged Ten Weeks, Operated on for Pyloric Stenosis and having received inadequate Saline Therapy.

	Alkali Reserve: Volumes of CO_2 per 100 Cubic Centimetres of Plasma.	Plasma Chloride Content: Milligrammes of NaCl per 100 Cubic Centimetres of Plasma.	Urinary Chloride Content: Percentage.	Hæmoglobin Value Percentage. (13·8 grammes = 100%.)	Erythrocytes: Millions per Cubic Millimetre.	Hæmatocrit Reading.
6/4/38	108	456	(trace)	85	4·6	35
7/4/38[1] [2]	92	482	0·01	86	4·6	35
20/4/38[2]	56	560	0·02	78	4·7	34

[1] These results were obtained after the child had received 180 cubic centimetres (six ounces) of normal saline solution by the intravenous route. The estimations were made twelve hours after the first ones.

[2] Operation was performed later during this day.

Estimations on discharge from hospital.

means of materially improving the clinical condition of the infants, of correcting the dehydration, and of restoring the state of alkalosis and the disordered chloride metabolism to an approximately normal condition.

6. As a result the operative risk for the infant is improved and a better post-operative course is ensured.

Acknowledgements.

I wish to express my acknowledgement to all members of the honorary and resident medical staff of the Children's Hospital for cooperation in this work. My thanks are especially due to Dr. J. G. Whitaker, Dr. Reginald Webster, Dr. H. Boyd Graham, Dr. V. L. Collins and M. A. Machie. Miss Green, of the biochemical section of the pathological department, has been most helpful; she has been responsible for all the biochemical estimations.

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THE MYCOTIC FLORA OF "SURFERS' FOOT" IN SYDNEY.

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The following account is based on two years' study of "surfers' foot" in Sydney. The material has come from the out-patient clinic at Royal Prince Alfred Hospital, from patients submitted to me by various colleagues, and from the physical examination of all applicants for employment at the Goodyear Tyre and Rubber Company's factory at Granville, as well as from the members of the Goodyear staff who have fallen victims of the fungi.

The name "surfers' foot" is unfortunate. Amongst the applicants for work at "Goodyear's", interdigital pedal tinea was found to be active in 38%; of these, just above one-half were not surfers; quite a few assured me they had not used public showers or bathing places.

Investigations overseas have revealed the presence of pathogenic fungi on new vegetable-tanned leathers, and it appears probable that the spread of the malady in Sydney during the last decade has been due to infected leather, and possibly still is.

The trouble is not known amongst fishermen, nor have I seen or heard of a patient under ten years of age, that is to say, during the "bare-foot" age for most of them, when not at school or out in "Sunday" clothes. It would seem that to go bare-foot is to avoid the malady, and since the incidence amongst women is less than amongst men, it is probable that the open shoes so fashionable with our women-folk permit greater ventilation and keep the interdigital spaces drier, thus protecting them from infection by the fungi.

In all, sixteen pathogenic fungi have been isolated and grown. These are distributed through the following genera: *Epidermophyton, Ectotrichophyton, Microsporum, "Monilia"* and *Eutorula*.

Of these, the epidermophytons were the most numerous, both in the number of species found and in the number of patients infected. Second in frequency of its occurrence was *"Monilia"*, and then *Eutorula*, represented by the recently described species *excorians* Kesteven[1] and then *Microsporum, Ectotrichophyton*. The lesions produced by these pathogenic fungi are all alike, and it has not been possible to predict which will be found. The only exception to this statement is that infections which were intractable, which failed to yield to the milder applications, proved in most instances to be due to a species of one of the hair-infesting genus *Ectotrichophyton* and/or to double infections. The clinical condition is too well known to need description, but a word about treatment may be permitted.

Most infections yield at once to a few applications of *Tinctura Iodidi Mitis*, or *Tinctura Iodidi Mitis, Tinctura Benzoini Composita* and glycerine in equal parts, or to forty-eight hours' continuous treatment with eusol. But if the lesion has healed up completely so that the skin looks perfectly clean and healthy, and if all subjective symptoms have gone, the condition may be far from cured. Either slowly growing mycelium or resting and resistant spores commonly remain and, given warm weather and moist conditions between the toes, the trouble will recur again and again. To obviate this it is necessary to apply the chosen lotion or paint at least once a week for a period of not less than three months.

Resistant recurring infection usually yields to drastic application of a 1% solution of dihydroxyanthranol ("Cignolin" or "Anthralin") in benzole. The best results are obtained if the feet are soaked in hot water for about twenty minutes, and if the skin between the toes and other infected areas is then scrubbed with a rough towel to remove as much of the horny layer as possible. The lotion should then be applied and rubbed in vigorously. If the reaction does not make the areas tender and painful, the procedure should be repeated in three days and again two days later. It should be repeated once a week for two to three months. Of course the patient should be tested against the lotion by its application to a small hairless area twenty-four hours before the general application; occasionally one meets patients who are very sensitive to

dihydroxyanthranol. Weaker solutions do not kill the fungus as efficiently as *Tinctura Iodidi Mitis*.

The form commonly known as *Tinea albicans*, in which extensive blebs or blisters are formed and the sensitive deeper layers of the skin are exposed, is best treated by the careful paring away of the dead skin and the application of a dressing of eusol-saturated gauze till the trouble settles down; the treatment may then be changed if found necessary.

Gentian violet solutions are commonly as effective as the other applications; but they are not such strong fungicides as the others, nor do they penetrate so deeply, and are therefore more likely to be disappointing if the fungus is one of the more resistant and/or more deeply growing forms.

Disinfection of shoes may be effectively carried out if they are shut up in formalin vapour for forty-eight hours. This may be done very simply in the following way. An ounce or two of formol or formalin (40% formaldehyde in water) is placed in a cup in the bottom of a tin large enough to hold the shoes as well (an ordinary biscuit tin serves admirably). These are held for a moment with the steam from a fast boiling kettle playing well up into the toes, and then placed in the tin, which is sealed by paper pasted round the overlap of the lid. The tin should be left sealed for forty-eight hours and, if possible, should be placed in a warm place to ensure the better vaporization of the formol. The shoes should be left in the sun for a day to ensure that the formol has been completely driven off before they are worn again. Formol is a very strong skin irritant.

Woollen socks may be sterilized in the same way without being damaged; others may be simply boiled.

TECHNIQUE.

Detection of the Fungi in Scales of Skin.

Two methods of staining the fungi were relied upon in the examination of skin scales or of hairs in other forms of infection: first, the clove-oil-phenol-picric-acid method described by myself last year,[2] and secondly, a modification of the method of Berberian.[3] This worker stained the pieces of tissue on the slide. I place them in a solid watch-glass, wash them in 90% and then in absolute alcohol followed by ether, each for three minutes at least. They are then hydrated. There is no need to come down the alcohol scale; they may be transferred from ether to alcohol of any strength above 50%. I usually use 90% alcohol and then transfer the pieces of tissue directly into water. They are then treated for one minute with glacial acetic acid. This is washed out with three changes of water and the scales are then covered with the stain of Martinotti, recommended by Berberian. They are left in this for not less than fifteen minutes, then washed with water and flooded with 1% acetic acid. In this they are left till no more colour comes away. They are now dehydrated with pure acetone. Alcohol must not be used after staining is completed, because it decolorizes the scales more or less completely. After being cleared in pure xylol the scales are mounted in Canada balsam for examination.

Not only fungi, but staphylococci and streptococci are stained a deep purple and stand out most strikingly. I regard the method as being absolutely reliable. The time spent in staining is saved in the examination, for the fungi are obvious if present.

The only confusion which arises is due to the fact that strands of mucin stain very similarly to the mycelial threads. These, however, never show septation, and when differentiated once or twice will not cause any doubt.

It may be noted here that both these staining methods fail completely to reveal "mosaic" fungus. On a number of occasions when mosaic fungus was observed in scales treated with potassium hydroxide, subsequent examination after these methods of staining were used failed to reveal a trace of the structure. I have formed the opinion that "mosaic" fungus is an artefact. I have never grown a pathogen from scales from a case in which other scales contained the "mosaic".

Culture Methods.

All the fungi were grown on Sabouraud glucose agar. The peptone used was Wittes's, and the glucose the dry powdered form put up by Elliotts and Australian Drug Proprietary, Limited, of Sydney. Descriptions are, unless stated to the contrary, all based upon the growth on plates kept at 35° C. The original scraps were plated out and examined every twenty-four hours. As soon as likely looking growths appeared portions were transferred to agar slopes and the plate growth was allowed to continue. If contamination appeared, as it usually did on the original plates, subcultures were made either from the plate or slope. Once growth was established and the fungus was identified as one of the pathogenic forms, the slopes were transferred to a cabinet and allowed to remain at room temperature. Drying of the slopes was prevented by immersion of the cotton wool plugs into sterile hard paraffin of melting point above 35° C.

For microscopic study portions of the growths were mounted in the cotton-blue-lactophenol solution and carefully ringed with white duco, which, being spread evenly over the whole of one end of the slide, also provided a firmly attached label.

It was also found valuable to take "impress" mounts. These were obtained as follows. A cover slip was treated with egg albumen solution[4] in as thin a film as possible, obtained by its being wiped practically clean with the bare finger; the cover slip was then firmly but gently pressed down on the surface of a colony. Care is taken to avoid any lateral movement, the cover slip is lifted up and after being dried completely it is flooded with glacial acetic acid, washed in water and dried again. It may now be stained. I have found Giemsa, Martinotti and 1% alcoholic gentian violet to give

excellent pictures. These "impress" preparations reveal the arrangement of the aleurospores in a manner which, whilst not absolutely correct, enables one to keep permanent comparative preparations.

It is freely admitted that, were one engaged upon a study of these fungi, *qua* fungi, to have done so confining oneself to their appearance upon a single culture medium would have been a very incomplete investigation. I am of the opinion, however, that for clinical purposes, which on the botanical side are purely taxonomic, the use of one single standard culture medium and method is not merely all sufficient, but desirable, as simplifying the procedure necessary for the identification of the numerous species.

When it was desired to bring pleomorphic colonies back to the fructifying form the following method was commonly successful. Pieces of hairless fœtal calf skin, collected and rapidly dried under aseptic conditions without being heated, were put into a Petri dish and just floated on 4% glucose solution. Tufts of the "wool" from the pleomorphic colony were planted onto the skin and incubated at 35° C.

Description of the Fungi Found.

The Trichophytonaceæ are particularly liable to pleomorphism. This makes their identification very difficult, and doubt commonly remains after a decision has been arrived at. In view of this fact the following descriptions are offered in the hope that, if the identifications are erroneous, the descriptions will serve to enable the necessary corrections to be made by later workers.

The members of the genus *Epidermophyton*[1] will be described first.

This genus presents two, at present, very clearly differentiated sections. On the one hand there are those forms typified by *Epidermophyton niveum* McCarthy, which, up to the present, have never been observed to develop closterospores, or *fuseaux* as Sabouraud descriptively designated them. On the other hand are those which do form these characteristic trichophytonid multiple spore cases.

The members of the genus found causing interdigital pedal tinea in Sydney belong for the most part to the *niveum* section of the genus. It should be recorded that, though described from glucose agar cultures, these *niveum* section species were grown on skin and other media in order that the closterospore stage might be obtained if possible.

Epidermophyton Niveum.

Epidermophyton Niveum McCarthy, Var. Closterosporiger, Var. Nov.

The *Epidermophyton niveum* McCarthy was described in the *Archives of Dermatology and Syphilology*, Volume VI, 1925.

The new variety *closterosporiger* is a rapidly growing species; it attains a diameter of 3·0μ in sixteen days, and then has a pure white opaque central area surrounded by successive zones of varying opacity; there is just a suggestion of radiating rays.

It is essentially similar to the *Epidermophyton niveum* of McCarthy, but in addition to the features of that species there are also present in the sixteen-day colony small closterospores. These are not typical; they are blunt at both ends, they are three to seven septate, each segment is as long as it is wide. Their diameter is around 6μ (actually 5μ to 8μ). They are identified as closterospores on account of their terminal situation, for otherwise they resemble short strings of chlamydospores.

In an old dry sixty-day colony the only spores found were large spherical chlamydospores, 10μ to 15μ in diameter. They appear to have been developed in different ways. The smaller stages may be observed as intercalary swellings, lateral buds and terminal swellings. There are none of the small aleurospores in the younger colony typical of *Epidermophyton niveum*. It is possible that these spores are formed by the enlargement of the smaller. Slopes fifteen months old are of a very light brown colour.

Epidermophyton Niveum McCarthy, Var. Coremiger, Var. Nov.

The new variety *coremiger* of *Epidermophyton niveum* McCarthy also resembles McCarthy's so closely that it appears unwise to describe it as a new species.

In colonies up to twenty-four days old the only spores seen were spherical aleurospores; but at twenty-four days intercalary chlamydospores were abundant. Coremia, suggesting the conidiophore of a small penicillium, were not uncommon. They present three to five short oval branchings of the thread, and these bear one to three aleurospores in series.

Mycelium has segments with a length of 2·8μ.

Aleurospores are oval, from 7·0μ to 10·0μ by 2·8μ to 3·0μ, and spherical 3·0μ in diameter.

Chlamydospores are spherical and have a maximum diameter of 5·6μ.

Basal segments of coremia are 11·3μ by 3·0μ.

Aleurospores are born clustered as lateral buds around the mycelial threads, as well as terminally on the coremia. A culture eighteen months old is quite white.

Epidermophyton Pedis Ota.

The *Epidermophyton pedis* Ota was described in *Bulletins de la Société de pathologie exotique*, Volume XV, 1922, as *Trichophyton pedis*.

A seven-day culture on Sabouraud's agar is pure white, circular, with a bun-like raised central area, but with a small central crater. The duvet is short, suggesting a woolly blotting paper. Later growths bear a longer duvet and are definitely woolly.

Up to ten days no terminal spores are visible; a few spirals and some intercalary chlamydospores are present.

[1] The nomenclature and classification adopted in this paper is that of Dodge.[3] He is the only thoroughly qualified botanist who has critically examined the pathogenic fungi, and I feel very emphatically that clinical mycologists will best serve the subject by accepting his guidance.

A thirty-day culture on Sabouraud maltose has invaded the whole of a 7·5 centimetre plate. Three varieties of area are present. First, there is a dense opaque white central area of blotting paper appearance; secondly, around this is a less opaque dusted white area; thirdly, there is what appears to be an unoccupied agar surface, but under the microscope this proves to be covered by a close reticulation of aerial and subcultural mycelium.

The under-surface of the opaque and less opaque areas is yellow, citron coloured to yellowish brown; the greater part of the apparently bare area is a sooty grey, and this colour is diffused through the medium.

The dense white opaque area of the growth is composed of a tangled mat of mycelium in which the hyphal threads are from 2·8μ to 4·2μ in diameter, and in which the meshes of the mat are more or less completely filled by the spores described below.

Their structure may be readily examined in both the dusted white opaque areas and the apparently bare area. The hyphæ are of the same average thickness, the septæ are very fine and far apart, so that until carefully examined the hyphæ appear to be non-septate.

A few chains of small thick-walled spores may be found interrupting the continuity of some of the hyphæ; these have been identified as the arthrospores.

Chlamydospores, large and globular, are exceedingly abundant and may apparently be developed either as intercalary, terminal or lateral spores. The young chlamydospore is oval, pyriform or fusiform. The fusiform form has an earlier, nearly cylindrical, stage. It first appears as a swollen segment of a mycelial thread; this gradually swells, becomes pyriform and finally globular. The lateral and terminal spores, which perhaps should be identified as aleurospores, are at first pyriform, gradually enlarge, and may pass through a fusiform stage before becoming globular. All may apparently be readily detached, and when they are free there is nothing to indicate whether they were originally intercalary or terminal. Early stages are 2·8μ by 3·5μ in the lateral and terminal forms. The cylindrical swelling of the early intercalary spore is commonly 12·0μ in length. The ultimate globular spore measures up to 14·0μ in diameter. No septation of any spore stage was seen.

Aleurospores are difficult to see in whole mounts, but in an "impress" preparation they are found to be quite abundant. They are oval, and the prevailing size is 1·6μ by 3·0μ, but larger, lighter staining examples are seen up to 3·0μ by 5·0μ. They are attached directly as lateral buds to the hyphæ, and compound branched sporophores were not seen.

A thirty-five-day culture on Sabouraud glucose at room temperature is six centimetres in diameter, with numerous satellite growths up to one centimetre in diameter. The growth is snow white and woolly, the underside light brown. In places, and especially on the satellite growths, the duvet is more open and has a coarser dusted appearance.

In these particular areas the mycelium is thicker and darker, and bears numerous arthrospores and many enlarged intercalary chlamydospores, these latter being from one to five and six celled. A few of these, terminally placed, closely resemble thick, blunt, round-ended closterospores. Such spirals as can be found are open. The greater mass of the mycelium is similar to the thicker portions, but finer and more translucent.

The mycelium is 1·4μ to 4·2μ in a fifty-seven-day culture. It is 2·8μ to 7·0μ in a thirty-five-day culture.

Intercalary chlamydospores in a thirty-five-day culture are up to 14·0μ by 9·8μ in diameter. Large hyaline chlamydospores of 14·0μ in diameter are very plentiful in a fifty-seven-day culture. The *"en raquette"* form of mycelium is common in a thirty-five-day culture.

A plant eleven months old is of a light coffee and milk colour with a tinge of red in it.

Epidermophyton Album, Sp. Nov.

On Sabouraud's glucose agar the growth of *Epidermophyton album* is snow white, quite opaque, and has a diameter of two centimetres in eight days. The centre is elevated to form a large prominent pimple; the extreme periphery is diaphanous. There are neither radial nor concentric furrows.

The mycelium is of medium thickness, with long segments. Neither closterospores, pectinate bodies, spirals, nodular or tangle bodies can be seen. Aleurospores are abundant, and their arrangement and size, relative to the mycelium, resemble closely Sabouraud's figure 362 of *Microsporon audouini*; but the mycelium does not display *"en raquette"* formation.

In a forty-four-day culture, large, fairly open spirals are common. Chlamydospores and arthrospores, both intercalary and terminal, are present, but closterospores cannot be found.

A fifty-day culture on Sabouraud's glucose agar completely covers a nine-centimetre plate. The central area of 2·5 centimetres is rose-tinted, flat and has a finely granular surface. Around this is an evenly raised zone 1·5 centimetres across. This is smooth and swells gradually to the thickest point in the centre of its width, and then as smoothly thins again. It is snow white. Outside this zone the growth is less dense and permits the brown tint of the medium to filter through. The under-surface is brown in the centre, fading to yellow.

A later subculture on maltose agar yielded a fine, smooth, elevated pleomorphic growth, quite devoid of characters.

The mycelial threads average 3·0μ in thickness, and their segments vary rather widely on each side of the common length, 25·0μ.

The aleurospores are oval or pyriform, 2·8μ by 3·5μ being the common size. They are attached directly to the hyphæ without pedicles either singly along the length or in terminal clusters. Branched sporophores were not seen.

The chlamydospores are spherical; the predominant size is 8·0μ in diameter, but both larger and

smaller examples are common. They are developed both as terminal and intercalary structures. Open spirals of three to five coils are not hard to find, but are by no means common.

Colonies two months old on slopes are rose-tinted. A plant twenty-one months old on glucose agar is pink; another of the same age on maltose agar is light cinnamon brown.

This new species resembles most closely *Epidermophyton planum*, to be described presently, but differs in the size and shape of the aleurospores, the possession of spirals and absence of typical closterospores.

Epidermophyton Flavum, Sp. Nov.

The original colony of *Epidermophyton flavum* growing around the skin piece is quite opaque and has a woolly blotting-paper like surface, pure white in colour, and has a diameter of two centimetres on the ninth day. The centre over and immediately around the piece of skin is heaped; the rest is smooth, but shows a tendency to zonation by more dense and thicker growth. The growing edge is perfectly even and diaphanous. The microscope reveals a moderately wide zone of growth beyond the diaphanous edge, and this is largely submerged, but not deeply so.

At this period, nine days' growth, the colony consists of rectilineal hyphæ, branching at acute angles and having a diameter predominantly 3·0μ, but varying slightly on both sides of that figure. Beyond an occasional club-shaped terminal swelling to a few of the threads, there is nothing in any way worth further mention.

A nine-day plant from this onto a Sabouraud's glucose agar slope has a length of 2·5 centimetres. It has a faintly yellow umbonate central area of 0·75 centimetre; the rest of the colony is pure white and woolly.

This colony at seventeen days has covered the whole of the exposed surface of the slope (six centimetres); most of the surface is faintly creamy and like blotting paper, with a dusted diaphanous marginal zone.

A three-day colony grown on Sabouraud's maltose has a diameter of 0·75 centimetre, is quite white, has a velvet surface, and is evenly heaped to form a tiny bun-shaped eminence. The reverse side has a coffee and milk colour.

In both the sixteen-day colony on glucose agar plant and in the eighteen-day colony on the agar slope the only spores are spherical aleurospores. These are abundant and apparently grow as lateral buds of any of the hyphæ, without visible sterigmata. They are oval, 5·0μ by 3·0μ in size.

In the central area of the eighteen-day colony open spirals of three to six coils are abundant. In this specimen occasional enlargements of the hyphal threads may be identified as intercalary chlamydospores or arthrospores.

The characters of the colony on Sabouraud agars are similar to those of *Epidermophyton niveum* McCarthy, but this species is not so dense in its growth and not so definitely zoned. The tinge of yellow also distinguishes it from *Epidermophyton niveum*, which it resembles in that the reverse side is brownish. From *Epidermophyton cerebreforme* Dodge it differs in that it is not cottony in the early stages, and in the absence of greying with age. From *Epidermophyton pedis* Ota it differs in the shape of the aleurospores, and the concolorous reverse. From *Epidermophyton gypseum* McCarthy it differs in colour and in the shape of the aleurospores.

It differs from all these in its yellow colour, in the absence of the chlamydospores, and in the presence of spirals, though these have been observed in *Epidermophyton cerebreforme* Dodge.

Cultures twelve months old have a greyish-yellow colour.

Epidermophyton Cerebreforme Dodge.

Epidermophyton cerebreforme was described by Dodge in his book "Medical Mycology", at page 479.

The growth of *Epidermophyton cerebreforme* on Sabouraud glucose agar is white and woolly. At the end of ten days three subcultures have diameters 1·5, 2·0 and 3·0 centimetres. There is a pinhead central nipple surrounded by a depressed blotting-paper like area, and outside this the growth is looser, deeper and woolly. The whole circular disk is opaque; the outer margin of the largest colony is slightly diaphanous. There are no radial striations or furrows.

Mycelial threads from this ten-day subculture are of two sorts. There are fine hyaline hyphæ, 2·5μ in diameter, and apparently non-septate. These are observed especially from around the periphery of the colony and from the surface wool. The second form of the hyphal threads is thicker, 3·5μ, granular and septate. Chlamydospores having a diameter of 7·0μ are formed along the length of the thicker hyphal threads, but are not numerous.

Aleurospores are common; they are small, having a diameter of 2·0μ by 2·5μ, and were observed only as lateral outgrowths from the thinner hyphal threads. They are oval or pyriform, and at times have a pointed end. They are very easily detached, so that it is not possible to assert that they do not grow in bunches or clusters, though none such were observed.

The microscopic characters were studied on material mounted in cotton blue lactophenol.

No closterospores, spirals or dendritic structures are present in the ten-day colony.

A thirteen-day colony on Sabouraud's glucose agar is circular and has a diameter of 5·0 centimetres. There is a central opaque, pure white umbo of 0·05 centimetre in diameter, and the reverse side of this is dark coffee brown. The rest of the colony is greyish-white, with a somewhat darker zone about 0·5 centimetre wide around the umbo. Beyond this zone the colony is zoned in five narrow bands and then a wider marginal zone. The whole surface is quite flat beyond the umbo, and presents a loose cottony surface; but there is very little depth.

Epidermophyton Macrosporicum, Sp. Nov.

Epidermophyton macrosporicum was first isolated from a case of tinea of the scalp, and the following description is based upon that strain.

On Sabouraud glucose medium one of the hairs was found to be sprouting a mycelium from the sheath near the end after forty-eight hours' incubation at 37° C. This was carefully lifted and transplanted to a new plate. At the end of seven days at 37° C. the growth has a diameter of 0·6 centimetre. It is quite flat and of a dusted appearance, without definite colour. Most of the growth is beneath the surface, but short terminal lengths are above the surface.

At this seven-day stage the mycelium is thick, thick-walled, and a great number of the threads bear terminal chains of short thick-walled arthrospores, with an occasional dilated thinner walled chlamydospore. There are no pectinate or spiral bodies and no closterospores present.

A sixteen-day culture is 1·5 centimetres in diameter. A heaped grey centre is partly surrounded by a narrow, opaque, grey area, and then the greater part of the growth is flat, diaphanous, faintly striated.

The mycelium is still as in the seven-day culture, but there are now developed very many dilated intercalary, terminal and lateral, thin-walled bodies.

A nineteen-day culture has a diameter of 2·0 centimetres. There is an opaque heaped umbo, a little zone 0·5 centimetre in diameter, a more translucent zone also just over 0·5 centimetre, then an opaque zone and a peripheral more diaphanous zone.

Branched sporophores carrying single pairs and threes of aleurospores at the ends of the branches are common; arthrospores of irregular shape and size are present but rare. Pectinate bodies and spirals are not seen.

Grown on Sabouraud's maltose agar, the plant in four days has a diameter of 2·0 centimetres. The thickest portion of the growth is at the centre, except for a small dimple; from its thickest the surface slopes evenly to the outer edge without any trace of zonation or folding. The colour is pure opaque white, with a narrow greyish growing margin. The surface resembles a very fine woolly blotting paper.

No spores of any kind are seen at this stage.

The seven-day plant presents very numerous elongated aleurospores. These average 3·0μ by 8·4μ, but vary slightly on both sides of the average.

In older cultures on glucose agar intercalary chlamydospores up to 28·0μ in diameter are very common, as also chains of arthrospores, averaging 8·0μ to 9·0μ in length and equally broad. As the plant grows older these spores become more and more abundant, and the aleurospores are no longer found. The chains of arthrospores develop into chains of large thick-walled chlamydospores along the length of the hyphæ. Many of the hyphæ terminate in swollen, shortly segmented ends, which suggest closterospores, but progressive stages show these to be chlamydospore formations. The end of these terminal segments is that they form terminal clusters of the chlamydospores.

The old colony becomes a mass of these large spores in a sponge-work of mycelial threads.

As the colony ages, the central area takes on a yellow tinge; otherwise it is a flat, opaque, white colony without ridges or zones, except for the semi-opaque growing margin.

A slope twelve months old is a dirty cinnamon brown in colour.

The very large size of the chlamydospores in old cultures differentiates this from every other species as yet described.

Group of Epidermophyton Floccosum.

Epidermophyton Interdigitale Priestley, Var. Rosea, Var. Nov.

Epidermophyton interdigitale was described by Priestley in THE MEDICAL JOURNAL OF AUSTRALIA of December 8, 1917.

The new variety, *rosea*, on Sabouraud medium in ten days has a circular growth which has a diameter of 3·5 centimetres. The woolly surface is pure white in younger stages, but gradually assumes a brown tinge centrally, and, moreover, the central area is blotting-paper like rather than woolly.

The mycelium is relatively fine, and its network presents a rectilinear angulated pattern, curved flexuous pieces being relatively few.

The only spores seen on the tenth day are aleurospores. These are arranged along mycelial filaments, either as simple series of lateral buds or in dense branching clusters, the latter being characteristic, but difficult to demonstrate in mounted specimens, because they are so readily set free.

Closterospores are rare. They are thin-walled, three to six loculate, tapering slowly to a thicker rounded distal end.

In a thirty-day growth the central area has a very light brown tinge; there is still no obvious duvet, the surface being blotting-paper like. Open spirals are common, but not numerous, and closterospores were not found. The growth planted on a slope on February 12 (66 days ago) has a faint brown tinge only. That plated out from this slope on April 25 (25 days ago) has a fairly extensive rose-pink central area, surrounded by an elevated woolly zone, then a dark line, an opaque white band, a brown band of poor growth, then a wider granular white band.

On Sabouraud maltose a forty-three-day colony almost covers an eight-centimetre plate. The surface is woolly and irregularly raised into low ridges and mounds. The colour is white, with the faintest tinge of brown over a large central area. The margin is wavy and presents a diaphanous edge. There is no extension beyond the growth beneath the surface. The mycelium is 1·6μ to 2·0μ in length. The aleurospores have a diameter of 3·0μ to 4·0μ, and are spherical to oval. The chlamydospores average 7·0μ in diameter and are spherical. The closterospores measure 42μ to 49μ by 7·0μ to 9·8μ.

An eight-day pleomorphic plant on maltose agar is snow white, with a long woolly duvet and a broad diaphanous cottony margin.

A slope twenty months old is a dirty grey in colour.

This differs from the typical form only in the colour of the plant on Sabouraud glucose agar; but it has been recovered from a number of patients and always presented the same characters, hence the subspecific designation.

Epidermophyton Planum, Sp. Nov.

On Sabouraud glucose a ten-day culture of *Epidermophyton planum* is quite circular and has a diameter of 4·5 centimetres. The woolly surface is flat, but with a more opaque, slightly raised, narrow ring near the periphery; beyond this the growth is flatter and more diaphanous.

The mycelium is rather fine and its segments are long. The septæ are not thick. Small aleurospores are very numerous and are borne singly on short pedicle-like segments on much-branched sporophores. The branching is not necessarily or even most frequently terminal; but clusters of the aleurospores are borne along the length of the mycelial threads.

Closterospores, though not rare by any means, are not very numerous. They are parallel-sided, and both ends are similarly blunt. They are easily detached, and when free the two ends are so similar that there is nothing to indicate which was the free end. The walls and septæ are thin. From three to six loculi can be found in various of the closterospores, which may develop singly or in bunches of as many as five.

The growth on Sabouraud maltose is essentially similar, but more woolly. The mycelium averages 5·0μ up to 7·4μ in length. The closterospores measure 8·4μ by 31·3μ. The aleurospores are spherical and have a diameter of 2·8μ to 3·0μ.

The great majority of the aleurospores are spherical, but there is a large minority which are pyriform, whose long axis is about one-third greater than the short—this averages 3·0μ.

A colony fifty days old completely covers a seven-centimetre plate. The centre has a woolly blotting-paper like appearance; the margin, slightly more woolly, is diaphanous. The whole surface is smooth, without folds of any kind. The centre is evenly lifted, so that the whole may be likened to a flat bun. The outer margin is quite regular, that is, without sinuation.

A culture eighteen months old is just sufficiently grey to prevent one calling it white.

All the closterospore-bearing epidermophytons heretofore described are pink in colour; at some stage of growth this remains white, and together with microscopic features this differentiates the species.

Epidermophyton Griseum, Sp. Nov.

The lesions from which *Epidermophyton griseum* was isolated were a patch of blisters on the sole of the right foot and a patch of papules with some exfoliation of the skin without actual excoriation on the outer and upper surface of the big toe. Unlike the other species, it has been isolated only once.

The fungus was grown on glucose agar from smears of the blister fluid (strain *A*) and from skin scraps plated out on maltose agar (strain *B*). The growth from the smears has been a unique occurrence; though it was often tried, this was the only successful attempt.

The plant on glucose agar has a diameter of 3·0 centimetres in four days; it has a definitely yellow centre with a surface slightly more woolly than blotting paper.

The centre of the colony carries numerous closterospores. These vary from 35·0μ to 60·0μ in length, 48·0μ to 52·0μ being the commonest length. The diameter is fairly constant at 6·0μ. The segments, usually five, may be as few as three and up to 6·0μ in length. There are no spines on these spores. The basal segment is hyaline; the terminal segment presents a blunt rounded end. For the most part they are borne singly on short lateral branches of the mycelial threads, but occasionally in pairs and not infrequently terminally.

At this early stage of the growth no other spores are present. The mycelial threads are of two kinds. Relatively straight threads are 3·0μ to 4·2μ in thickness, and fine, sinuous, less granular threads are from 1·5μ to 2·3μ thick. Large open tangle bodies composed of these finer threads are relatively common.

A day or two later aleurospores are present and are exceedingly numerous. These are spherical, have a diameter of 2·5μ, and are borne in small dense clusters on lateral sporophores; also terminal compound thryses are very numerous.

The tangle bodies break up into dense clusters and chains of spherical arthrospores, having a diameter of 3·5μ.

Chlamydospores were not seen.

Once the aleurospore formation commences, closterospore formation ceases; so that in a colony of six to seven days' growth these early spores are completely hidden where they lie in the centre and are not present in the outer zones.

The form undergoes pleomorphism very rapidly, and self-planted colonies around the parent present nothing but characterless mycelial threads.

A thirteen-day plant covers a seven-centimetre plate. It is grey, concentrically zoned, with many fine radiating ridges.

The under-surface is coffee and milk brown centrally at four days, and the colour gradually spreads.

On glucose the plant is always grey for the first few weeks, but turns a dusky rose-pink with age; slopes twelve months old are of that colour.

On maltose agar the growth is more woolly and is coloured citron-yellow, but is similar in microscopic characters.

Ectotrichophyton Mentagraphites Robin, Var. Chibaense Ogata.

Ectotrichophyton mentagraphites was described by Robin. The variety *chibaense* was described by Ogata in the *Japanese Journal of Dermatology and Urology*, Volume XXIX, 1929. This variety, grown on Sabouraud's glucose agar, is very similar in microscopic appearance to *Epidermophyton cerebreforme* Dodge. Since its first recognition it has been isolated twice. In all three cases the infection was very resistant to treatment, and it is probable that two patients, similarly difficult to treat, who were seen prior to the differentiation of the two species, and who were regarded as being infected by *Epidermophyton cerebreforme*, were really infected with this species. As will be seen, the microscopic characters are quite different.

A ten-day culture on glucose agar slope is the full width of the surface and 3·0 centimetres in length. The surface is blotting-paper like, and just a little more velvety than *Epidermophyton cerebreforme*. It has the same very faint tinge of yellow; but may, like that, be described as white. It is at the three to four day growth stage that the two may be recognized. At that age this has a more open cotton-wool like surface, which appears as though dusted with minute particles of white chalk when examined against the dark surface. The reverse side is yellow.

In the ten-day culture pyriform aleurospores, 2·5μ by 3·5μ, are very numerous. They grow closely clustered along straight unbranched mycelial threads, and also grow in compound terminal thryses, which are very numerous. Each aleurospore of those borne on the straight threads appears to be borne on a pedicle, which is very constantly 7·0μ in length and nearly as thick as the spore. These are as readily set free, when being mounted for microscopic study, as the spores. Therefore there appear to be two forms of spores present and in equal abundance.

In the thirty-day culture large spirals are very numerous. Their form is variable, from the nearly flat watch-spring form to that of the corkscrew.

Closterospores are exceedingly hard to find. The few seen have four to six segments, taper from a blunt, rounded, free end to the basal truncated segment. Their diameters are from 40·0μ to 55·0μ.

Ogata failed to mention closterospores. In commenting on this fact Dodge (page 506) states that they are probably present.

A culture nine months old has a pinkish, dusty grey colour.

Microsporum Audouini Gruby and Microsporum Canis Bodin.

Microsporum Audouini was described by Gruby in *Comptes rendus hebdomadaires des séances de l'Académie des sciences*, and *Microsporum canis* was described by Bodin in 1902.

Thanks to the kindness of Dr. N. F. Conant, who sent me subcultures of all the species he described in his recent paper,[6] I have been able to make absolutely certain by direct comparisons that these two species are correctly identified. In view of Conant's careful detailed description there is no need to do more here than to record their having been found.

Microsporum canis is one of the fungi resistant to treatment when it infects the interdigital spaces in the foot.

This completes the list of Trichophytonaceæ found up to the present time causing pedal tinea in Sydney. Before turning to a few miscellaneous pathogenic fungi from other families it might be well to warn readers that if they miss from the above list specific names they might have expected to find, the explanation will probably be found by reference to Dodge's "Medical Mycology". He found it necessary under the rules of botanical nomenclature to replace a few well-known names by others less known.

Miscellaneous Fungi.

Syringospora Albicans Robin.

Syringospora albicans was described by Robin in 1853.

Syringospora albicans is the familiar *Monilia albicans* under the name which Dodge has adopted. It was identified by comparison with the British National Type under the name of *Candida vulgaris*.

Candida Krusei Castellani.

Candida Krusei was described by Castellani in the *Philippine Journal of Science* in 1910.

It was identified by me by comparison with a subculture received from Sir A. Castellani. This is another of the forms which have been known in the past as "monilias".

These two "monilias" are of common occurrence, and one or other is the usual cause of malodorous soft skin between the toes. I have seen no case of bleb formation or excoriation due to the presence of these organisms alone. Always, if they are present when excoriation, deep exfoliation or bleb formation has occurred, then either one of the Trichophytonaceæ, *Staphylococcus aureus* or streptococcus also has been isolated.

On the other hand, chronic pedal paronychial infections from which one of the monilias alone is isolated is not uncommonly exfoliative and deep-seated.

Eutorula Excorians Kesteven.

Eutorula excorians was described by me in THE MEDICAL JOURNAL OF AUSTRALIA of June 4, 1938. I have isolated *Eutorula excorians* a number of times since the descriptive paper left my hands; it is capable of producing excoriation between the toes without the assistance of any other organism.

Cepholosporium, Sp.

Dodge states of the genus *Cephalosporium*:

Several species have been reported as pathogenic, but the group is mostly saprophytic, and reports should be scrutinized with extreme care to see that pathogenicity has been proved. They are very frequently found as contaminants in laboratory cultures.

I have isolated what is apparently the same species on a number of occasions, twice from every skin scrap plated out. On no occasion was it found in pure culture, but was always accompanied by another pathogen; yet I have formed the opinion that it increases the pathogenicity of the other fungus. In a number of instances when it has been present, the degree of desquamation, excoriation and irritation has been more severe than would have been expected from the pathogen present, and the condition has been more than ordinarily resistant to treatment.

I have had one unfortunate, or fortunate, depending on the point of view, opportunity of studying this fungus.

In the late summer of 1936 my own left foot developed interdigital tinea with some excoriation. *Syringospora albicans* was isolated, and the condition cleared up after a few applications of *Tinctura Iodidi Mitis*. A *Cephalosporium* species was also found on the plate, but was disregarded.

In the early summer of 1936-1937 the condition suddenly flared up again. This time the trouble affected all the interdigital spaces of the right foot and extended up onto the dorsum for some distance. The whole area was very hyperæmic, intensely irritating, with numerous small vesicles. Again *Syringospora albicans* and the *Cephalosporium* species were present on the plates, and both grew from every scrap. Mycelial threads were present in the scales. No *Trichophyton* was found. The trouble proved most intractable and did not clear up until drastically treated with the 1% solution of "Cignolin" in benzole.

I have since seen a similar condition in which the offending organisms were the same, and another in which the *Cephalosporium* was accompanied by *Epidermophyton flavum*. This last, as a rule, responds to quite mild applications.

Of the species listed and briefly described by Dodge this resembles most closely *Cephalosporium niveolanosum* Benedek.

Conclusion.

This completes the list of fungi, definitely or very probably pathogenic, which have so far been isolated from patients suffering from "surfers' foot". But, in addition, some few aspergilli and two as yet unidentified yeast-like forms are under suspicion owing to their presence, without accompanying pathogens, in a number of lesions which are clinically pedal tinea. These were all of them rather severe conditions, for which rapid cures were not found, so that I have not had the courage to experiment with them either on myself or on volunteers. Later, perhaps, I may have the opportunity of testing them on animals.

As the two stains used are not very well known, I append their formulæ.

Cotton Blue Lactophenol:

Phenol crystals	20%
Acid lactic (syrupy)	20%
Glycerine	40%
Aqua Distillata	75·0 cubic centimetres

Mix and add 0·5% methyl blue.

Martinotti's Stain:

Toluidin blue	1·0 gramme
Lithium carbonate	0·5 gramme
Aqua Distillata	75·0 cubic centimetres

Dissolve the dye completely and then add:

Glycerine	20·0 cubic centimetres
Alcohol, 95%	5·0 cubic centimetres

Acknowledgements.

I have gratefully to thank the directorate of Goodyear in Australia for the whole of my laboratory equipment and supplies. I should also like to thank my colleagues generally, and especially Dr. J. C. Belisario, Dr. A. L. Dawson, Dr. F. C. Florance, Dr. G. Lindeman, Dr. E. H. Molesworth and Dr. C. F. Robinson, of the Royal Prince Alfred Hospital clinic, for the opportunity to study and report upon their patients. I have to thank Sir Aldo Castellani and also Dr. N. F. Conant, of Duke University, Durham, North Carolina, who have both sent me type subcultures, which have enabled me me to make some identifications with complete confidence. To Dr. C. W. Dodge, of Missouri Botanical Gardens, and to Dr. W. C. Sawers, of the School of Tropical Medicine, University of Sydney, I am indebted for assistance in my work.

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FRACTURES OF THE OS CALCIS.[1]

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In spite of all the work that has been done in recent years on fractures of the *os calcis*, they are still the most difficult of all the fractures of the lower extremity to treat satisfactorily. In spite of treatment by the most approved methods, they are productive of more permanent economic disability than other injuries of this extremity. Böhler classifies these fractures into eight groups. Many of these groups comprise cases of considerable rarity, and I propose to confine myself to the common types produced by the typical form of violence.

Fractures of the type under consideration are almost invariably the result of the same type of violence: the patient falls from a height and lands on his feet. Thus bilateral fractures are very

[1] Read at the annual meeting of the Australian Orthopædic Association (British Medical Association), March, 1938.

common. In nearly every case the patient has lost his balance, knows that he is certain to fall, and jumps deliberately with a view to landing on his feet. Many of the patients are painters and tilers and others who are accustomed to work in thin rubber-soled shoes, which offer no protection to the heel when the fall occurs. The force applied is compression, and the astragalus is driven into the *os calcis*, the fracture being essentially intra-articular. Though there are many variations of this fracture, the common type of fracture with displacement is remarkably constant. Generally there is considerable comminution, the bone is shortened and widened, the outer wall of the *os calcis* is broken off and displaced outwards, and this displacement is usually well seen in an axial X ray picture. Persistence of this displacement is apt to cause considerable pain, owing to impingement of the tip of the external malleolus on the outer surface of the *os calcis*.

Another important feature is the breaking up of the upper articular surface of the *os calcis*; frequently portion of its posterior articular facet is actually driven into the substance of the bone and is quite detectable in a good lateral X ray picture.

The third important displacement is the angulation and displacement upwards of the tuber, as evidenced by alteration in the so-called tuberosity-joint angle. This angle should always be carefully inspected in the lateral X ray picture. It is measured by the intersection of two lines, one drawn along the upper non-articular part of the bone, and the other from the normally highest point to the anterior angle. The intersection of these lines as a rule makes an angle of 35° to 40°, but in such a fracture the tuberosity is often drawn so far upwards as to be at a level higher than that at which the highest point of this bone is normally situated. Often the astragalus appears to be actually embedded in the *os calcis*, and its long axis, instead of being oblique, becomes horizontal. The alteration of the axis of the astragalus results in subluxation of the mid-tarsal joint, the cuboid also becoming displaced downwards.

Another feature that is apt to be overlooked is the not infrequent adduction of the posterior part of the *os calcis*, which, if uncorrected, results in considerable pain on walking. The so-called traumatic flat-foot following these fractures differs very greatly from the ordinary static flat-foot in which the heel is everted and the astragalus is more oblique in its long axis than in the normal foot. At least three X ray pictures of the injured foot must be taken, the lateral and the oblique views being the more important.

Only in comparatively recent years in most clinics was any attempt made to reduce these fractures. The part was merely fixed in plaster with the deformity unreduced, and weight-bearing was prohibited for varying periods. The results were almost uniformly bad. I learn with surprise that quite recently some members of the Liverpool school have abandoned attempts at reduction by compression and traction and have reverted to the former policy of inaction. I learn with greater surprise that they express themselves as satisfied with their results.

It is rare to secure a perfectly satisfactory functional result from a typical comminuted fracture of the *os calcis*; but it is certainly true that the more nearly the shape of the bone is restored to normal, the closer will subsequent function approach to normal.

The three main objects to be attempted in carrying out reduction are: (*a*) to reduce the width of the broadened bone, (*b*) to pull down the tuberosity from its elevated position, and (*c*) to open up the joint space between the astragalus and the *os calcis*. Many people advocate waiting for the swelling to subside before reduction is attempted; but here, as elsewhere in the body, the principle holds good that the earlier the reduction of a fracture is attempted, the less difficulty will it offer, and I can see no good purpose in delay. It has become apparent that only continuous traction will prevent redisplacement of the fragments by the pull of the *tendo Achillis*. My own experience has been that transfixion by means of pins through the tibia and the heel is much more efficacious than continuous weight traction applied by means of a pin or hook in the heel alone. It is advisable to place the pin in the *os calcis* as high up and as far back as possible and to use a pin of small diameter in the tibia. This pin must not be placed too near the crest for fear of its splitting the bone. After powerful traction has been made in the manner described by Böhler, the bone is compressed by the clamp, and an unpadded plaster is applied with the pins remaining in position.

I usually rest the limb in a Thomas bed knee splint bent at the knee. I have as yet had no difficulty with the circulation and have never found it necessary to split the plaster, nor have I myself encountered any infection about the site of insertion of the pins, though I have seen several cases at a late date in which such infection had occurred. I do not know whether plaster of Paris is sterile, but I have certainly never met with infection in the presence of pins or hooks protruding through plaster or in wounds accidentally made whilst a plaster was being split.

Böhler apparently allows the patient to walk with the addition of a walking iron and with the pins *in situ* one week after reduction. I feel strongly that this procedure must be responsible for some infections about the pins, and occasionally for redisplacement of the fragments. Böhler leaves the pins in position often for ten to fourteen weeks.

The whole history of the treatment of these patients reveals the strong liability of the fragments to return to their former displacement, and I now prohibit all weight-bearing for a period of four months after reduction. I am convinced that since I have followed this procedure the maintenance of reduction and the final functional result have been better. In the convalescent period the use of an

Unna's paste bandage, an arch support and altered boots are of advantage.

If we consider the ultimate functional results, all cases must be differentiated into two main groups: those in which the articular surface of the bone is not fractured, and those in which this area is damaged and displaced. The former group usually gives a good result; the latter and commoner group must invariably lead to some limitation of function and carry the same more serious prognosis as any intraarticular fracture involving a weight-bearing joint, with its accompanying sequelæ of roughening of joint surfaces, limitation of joint movements and traumatic arthritis. The very nature of the fracture precludes me from sharing Böhler's optimism as to the recovery of full working capacity. These fractures in their prognosis are parallel to the comminuted fractures involving the lower articular surface of the tibia. My experience of these typical cases involving not merely broadening of the bone, but comminution of the articular surface, is that they are invariably followed by limitation of movement, particularly of subastragaloid joint movement, and fatigue on prolonged exertion. It is rarely safe to allow these men to resume work in the building trade if they have to work in dangerous situations, where lack of sure-footedness may cause a serious fall. On the other hand, they mostly become capable of remaining on their feet for long periods of the day, provided that their work is restricted to standing and walking mainly on level surfaces. Waiters and shop assistants are usually restored to full earning capacity; but, unfortunately, this accident is mostly sustained by those engaged in the building trade, and many of them never become capable of resuming their previous occupation.

When persistent disability remains two main operative procedures have at times been carried out. The first of these aims at removing pain caused when the fracture is unreduced by impingement of the lateral malleolus against the broadened *os calcis*; it consists of chiselling away portion of the lateral surface of the bone so that two surfaces no longer make contact with one another. This is occasionally effective, but is seldom applicable, as with this broadening there usually coexist fracture and displacement of the articular surface, and arthritic changes subsequently take place in the subastragaloid joint; all this is responsible for the major part of the disability, so that the operation succeeds in removing only the lesser factor.

To overcome this main source of pain, arthrodesis of the subastragaloid joint is sometimes carried out, but the results are often disappointing. We all know that stabilizing operations on the foot in anterior poliomyelitis are most successful when carried out in childhood, but that when performed in adult life they too often yield a good anatomical but a poor functional result. As is to be expected, the same disappointment is met with when arthrodesis of the subastragaloid joint is performed as a late treatment in malunited fractures of the *os calcis*.

THE CAUSE AND PREVENTION OF BLINDNESS IN TASMANIA: A SUPPLEMENTARY REPORT.[1]

By J. BRUCE HAMILTON and W. D. COUNSELL,
Hobart.

THE report prepared for and presented to the Interstate Conference of Blind Institutions held at Hobart in February, 1937, was the most complete review of blindness in Tasmania that it was possible to make up to December 1, 1936. The report was also presented to the first session of the National Health and Medical Research Council, held in Hobart from February 1 to 3, 1937, and the following recommendations were made to that council:

> That, as at least 40% of the blindness in Tasmania (and probably in the rest of the Commonwealth of Australia) is preventible, active measures should be taken by the Commonwealth Government to lessen its incidence. We propose the following measures should be adopted:
>
> (*a*) That voluntary sterilization should be available for carriers and sufferers from hereditary eye diseases, and that consanguineous marriages should be entirely prohibited.
>
> (*b*) That arrangements should be made for voluntary premarital certification.
>
> (*c*) That only certified persons should be allowed to use explosives.
>
> (*d*) That the eyes of artisans in certain industries should receive compulsory protection.
>
> (*e*) That the midwives' regulations should compel the notification of any abnormal condition in the eyes of infants under the age of twenty-eight days, and should incorporate precise instructions for the adequate prophylaxis of *ophthalmia neonatorum*.
>
> (*f*) That all pregnant women should be compelled to undergo adequate antenatal supervision.
>
> (*g*) That existing Commonwealth regulations should be more rigidly enforced to prohibit the landing of immigrants with signs of active or latent trachoma, and that trachoma should be a notifiable disease throughout the Commonwealth.
>
> (*h*) That only registered medical practitioners should be permitted to treat diseases of the eye, including refractive errors in childhood.
>
> (*i*) That sight-saving classes should be established in every city of the Commonwealth with a school population of over 5,000.
>
> (*j*) That routine examinations of the eyes of school children should be carried out by school medical officers at least once yearly, and that there should be provision for expert treatment and adequate follow-up.
>
> (*k*) That the services of an ophthalmic surgeon should be available to the inmates of all charitable institutions.
>
> (*l*) That the registration of the blind should be compulsory.
>
> (*m*) That the Commonwealth Government should adopt provisionally the model form of certification recommended by the English Committee for the Prevention of Blindness (Evans, 1936) until a standard British form is provided.
>
> (*n*) That the certification of blindness should be permitted only by medical practitioners possessing special knowledge in ophthalmology.

[1] This work was carried out with the assistance of a grant given by the National Health and Medical Research Council, which is gratefully acknowledged.

SUPPLEMENTARY DATA TO JUNE 1, 1938.

On February 5, 1938, we received a request from the Tasmanian Branch of the British Medical Association that our report should be brought up to date, and the following is a complete supplementary survey of blindness in Tasmania up to June 1, 1938; and, although we have found an increased number of blind, namely 195, we have not materially altered our previous recommendations and analysis.

Deaths.

Between December 1, 1936, and June 1, 1938, at least five of the 170 reported blind have died. Of these, only four were certified, and we were able to obtain only unreliable clinical information of the fifth. Of the four about whom evidence was reliable, two suffered from optic atrophy (the condition of one of these was supposed to be hereditary), the third was suffering from sympathetic ophthalmia, and the fourth from a pigmented growth of the eye (possibly a sarcoma) followed by secondary deposits in the liver.

New Cases.

With these five deaths our total blind of Tasmania as recorded in our original paper was reduced to 165. During the last eighteen months we have been able to find another 30 blind persons in Tasmania. Eleven of these thirty have been certified for blind pension purposes. Out of these eleven, four have been certified by ophthalmic surgeons and seven by general practitioners who, as far as could be ascertained, had not sought expert advice before certifying the patients. One of these seven is intimately known to us, and therefore, of the eleven certified, the clinical information is reliable in five cases.

Certified Cases with Reliable Clinical Information.

The five persons who have been certified as suffering from blindness and about whom reliable clinical information is available, suffer from the following visual defects:

Case I.—Detachment of the retina in one eye, and cataract in the other.

Case II.—Macular degeneration, right and left.

Case III.—*Retinitis pigmentosa.*

Case IV.—Iridocyclitis and secondary glaucoma.

Case V.—Interstitial keratitis and secondary glaucoma.

Certified Cases with Unreliable Clinical Information.

In the six cases in which, in our opinion, the clinical information was unreliable, the following diagnoses have been made:

Case I.—Right eye, corneal opacities; left eye, blind, cause uncertain.

Case II.—Optic atrophy, cause not stated.

Case III.—"Blinded." (No indication was given of the cause of blindness.)

Case IV.—"Blind." (No indication was given as to the cause of blindness.)

Case V.—Cataract, right and left. The patient was fifty years of age.

Case VI.—Right eye, totally blind. The cause was not stated. Left eye, corneal opacities, cause doubtful.

Uncertified Cases with Reliable Clinical Information.

There were nineteen patients, known to one of us, concerning whom the clinical information was entirely reliable. To the best of our knowledge not one of these is yet receiving a pension. They were suffering from the following conditions:

Case I.—Bilateral papillitis, possibly due to vitamin deficiency.

Cases II and III.—Thrombosis of the central veins in cases of hyperpiesia.

Cases IV and V.—High myopia with secondary macular defects.

Case VI.—Leucoma in the left eye and senile cataract in the right. Operation was refused by this patient.

Cases VII to X.—Iridocyclitis. In one case the condition was post-operative, in the second the condition was due to injury, and in two the ætiology was obscure.

Cases XI to XIII.—Optic atrophy. In two cases the condition was due to syphilis, in the third it was possibly due to birth injury.

Cases XIV to XVIII.—Primary glaucoma.

Case XIX.—Senile cataracts in both eyes. The patient refuses operation.

With the above details in hand, Table I, at page 60 of "Causes of Blindness in Tasmania, 1936", has been brought up to date and is published herewith. This table gives an analysis of the 195 patients under review.

Statistical Survey.

In the first annual report of the Tasmanian Institution for the Blind, 1888, it is noted that there were 180 persons blind in Tasmania, and at that date the population of the State was 136,700, making a ratio of 131·6 blind per 100,000 of population.

In 1936 we found 170 blind in Tasmania with a population of 230,000, approximately 74 per 100,000 of the population. Today we find 195 blind in Tasmania with a population of 235,527, or approximately 82·8 per 100,000.

Recommendations.

The recommendations printed at the beginning of the supplement delivered to the National Health and Medical Research Council some eighteen months ago still stand in the light of this new survey; yet we have no information that action has been taken upon any of them, and we should again like to lay particular emphasis on two or three points.

Adequate Certification and Complete Certification Forms.

Of the eleven patients certified during the last eighteen months only four were certified by ophthalmic surgeons, with the result that this survey is made more difficult and less effective, and also adequate scope is left for pensions abuse. It is not difficult to imagine that malingerers can outwit the general practitioner and cost the Commonwealth and the State a considerable sum in their life-time. One of us during the last six months has seen three patients, not blind within the meaning of the Commonwealth Act, who were sent for certification

Disease.

Group.	Anophthalmos. ? Cause.	Cataract—Congenital. (p)	Cataract—Senile. (p and h)	Cataract—Traumatic. (i)	Choroiditis. ? Cause.	Detachment of the Retina. Hereditary. (p and h)	Detachment of the Retina. Idiopathic.	Eale's Disease.	Glaucoma. Primary. (p)	Glioma.	Injury. (i)	Injury—Optic Atrophy. (i)
I. Receiving pensions—	2	16	3	1	2	1	1	1	6		13	1
(a) Blind — reliable clinical information	2	15		1	2	1	1	1	5		7	1
(b) Blind—unreliable clinical information			3						1		6	
(c) Not now blind—reliable clinical information ..		1										
II. Not receiving pensions—		6	4			1			5	1	4	1
(a) Blind — reliable clinical information		4	2			1			5	1		1
(b) Blind—unreliable clinical information		2	2								4	
Totals IA and IIA. Blind—reliable clinical information	2	19	2	1	2	2	1	1	10	1	7	2
Total IB and IIB. Blind — unreliable clinical information		2	5						1		10	
Grand totals IA and IB, IIA and IIB	2	21	7	1	2	2	1	1	11	1	17	2

Group.	Injury—*Phthisis bulbi*. (i)	Injury—Sympathetic Ophthalmia. (p and i)	Interstitial Keratitis. (p and v)	Iridocyclitis. ? Cause.	Leucoma. ? Cause.	Myopia. (h)	Neuroretinitis. ? Cause.	Nystagmus—Congenital. (p and h)	*Ophthalmia neonatorum*. (p and v)	Optic Atrophy. ? Cause.	Optic Atrophy—Hereditary. (Leber's Disease.) (p and h)	Optic Atrophy—Post Neuritic.
I. Receiving pensions—	2	5	3	10	2	9	2	1	12	8	9	3
(a) Blind — reliable clinical information	2	5	3	9	0	4		1	7	2	8	2
(b) Blind—unreliable clinical information				1	2	5	1		5	6	1	1
(c) Not now blind—reliable clinical information ..							1					
II. Not receiving pensions—				4		2	1			3	1	
(a) Blind — reliable clinical information				4		2	1			1	1	
(b) Blind—unreliable clinical information										2		
Totals IA and IIA. Blind—reliable clinical information	2	5	3	13		6	1	1	7	3	9	2
Total IB and IIB. Blind — unreliable clinical information				1	2	5	1		5	8	1	1
Grand totals IA and IB, IIA and IIB	2	5	3	14	2	11	2	1	12	11	10	3

Group.	Optic Atrophy—Tabetic. (p and v)	Optic Atrophy—Whooping Cough.	*Retinitis pigmentosa*. (p and h)	Thrombosis of the Central Vein.	Trachoma. (p)	Unclassified.	Total.	Preventible. (p)	Hereditary. (h)	Injury. (i)	Venereal Disease. (v)
I. Receiving pensions—	2	1	5	0	2	18	141	65	28	22	17
(a) Blind — reliable clinical information	2	1	4	0	1		87	52	18	16	12
(b) Blind—unreliable clinical information			1	0	1	18	52	12	10	6	5
(c) Not now blind—reliable clinical information ..							2	1			
II. Not receiving pensions—	2			2		19	56	19	8	5	2
(a) Blind — reliable clinical information	2			2			27	15	6	1	2
(b) Blind—unreliable clinical information						19	29	4	2	4	0
Totals IA and IIA. Blind—reliable clinical information	4	1	4	2	1		114	67	24	17	14
Total IB and IIB. Blind — unreliable clinical information			1		1		81	16	12	10	5
Grand totals IA and IB, IIA and IIB	4	1	5	2	2	37	195	83	36	27	19

by the Pensions Department. At least two of these were capable of deceiving a practitioner inexperienced in ophthalmology.

We would still emphasize the fact that the certification forms are wholly inadequate for diagnostic and statistical purposes, and while their use is continued unsatisfactory returns must result.

Propaganda.

The heavy incidence of patients blinded by chronic glaucoma shown by this report is most deplorable. This forces us to the conclusion that a great deal more information could be given to the public concerning the signs and symptoms of impending blindness and the need for expert attention. It seems to us that the need for a national society for the prevention of blindness in Australia is even more apparent than it was eighteen months ago. Since we have completed this supplementary list two further cases of long-neglected chronic glaucoma have come under our notice, and both persons are now certifiable as blind. In other countries much attention has been given to the preventive aspect, especially in America, where an enormous amount of work has been done by the National Society for the Prevention of Blindness. This body, whose headquarters are in New York, has, by means of lectures, pamphlets, films and broadcasting, done much to enlighten the populace of the United States as to the dangers of ocular neglect, and a similar body in Australia is more than justified.

Sight-Saving Classes.

The necessity for the institution of sight-saving classes in Tasmania is still apparent, there being two new cases of blindness from high myopia. In one of these, if defective vision had been corrected along the right lines, blindness might have been prevented. In the year 1938 no less than 571 sight-saving classes were open in the United States, and we know that in San Francisco such a class has been opened solely for the care of the Chinese children suffering from defective sight.

Venereal Disease.

There are two new cases of tabetic optic atrophy. In the previous

report two cases of tabetic optic atrophy were recorded; these further two cases make a total of four and bring the percentage of blindness from syphilitic optic atrophy to just over 2% of the total of 195. We are of the opinion that the treatment of syphilis in Tasmania is still very inadequate and, especially in the light of recent advances in hyperthermic therapy, we feel that more attention should be given to the individual patient and that a rigid system for the detection and surveillance of the defaulters in treatment should be established. We have frequently encountered patients who gave up treatment simply because they did not like it. In many cases the patients had no idea of the risk they ran; no duress had been used and no reminder had been given to induce them to continue treatment.

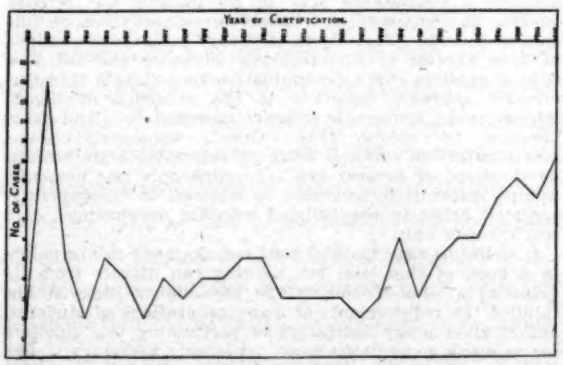
When referring to venereal disease it is interesting to note that of thirty new cases of blindness none is due to *ophthalmia neonatorum*, and there are still no children in the Tasmanian Institute for the Blind receiving education for defective sight due to this condition. We believe that this must be attributable to the more general use by the medical and nursing professions of adequate prophylactic measures.

Pensions and Curable Blindness.

Consideration should be given to the desirability of withholding pensions from blind persons who refuse medical or surgical treatment when in the opinion of the certifying ophthalmic surgeon such treatment might relieve their blindness.

Conclusion.

The accompanying graph illustrates the steady yearly increase of certified blind pensioners in Tasmania since 1930, and at least illustrates one point, and that is the growing cost of blindness to the Commonwealth Government. If for no other



reason than this, the problem of blindness in Australia should be immediately and exhaustively investigated. The application of the suggestions in this report should almost halve the prevailing incidence of blindness in the Commonwealth.

Acknowledgements.

We should again like to thank the Acting Deputy Commissioner of Pensions in Tasmania, Mr. A. F. Reisz, for his courteous assistance, and to acknowledge the financial aid generously given by the Commonwealth National Health and Medical Research Council.

Reports of Cases.

SUCCESSFUL TREATMENT OF PNEUMOCOCCAL MENINGITIS WITH "SOLUSEPTASINE" AND "M & B 693".

By H. E. Robinson, M.B., B.S.,

Honorary Eye, Ear, Nose and Throat Surgeon, Bendigo Base Hospital, Victoria.

Clinical History.

J. McK., single, aged thirty-nine years, was referred to me from Dr. May, of Barham, on December 16, 1938. The patient had been "off colour" for a few days, and for the previous two days had complained of right earache. On the afternoon of December 16, 1938, his temperature rose to 39·4° C. (103° F.), headache became severe and his neck became stiff. On his admission to hospital that night his temperature was 37·5° C. (99·6° F.), his pulse rate was very irregular, varying between 60 and 80 in the minute. The patient was irrational at times and called out frequently. His headache was intense, the neck was rigid and Kernig's sign was present. The right *membrana tympani* was a little red and full in the attic region, otherwise it appeared normal. No mastoid tenderness could be elicited.

At midnight on December 16, 1938, lumbar puncture was performed under general anæsthesia and 25 cubic centimetres of very turbid fluid were obtained under increased pressure. Five cubic centimetres of 10% "Soluseptasine" were injected intrathecally. Paracentesis of the right drum was then performed, and a small quantity of thin fluid was seen to escape. Very little was found in the bone when the mastoid was opened. A large area of the dura of the middle fossa was exposed, and the wound was then packed with acriflavine emulsion.

The cerebro-spinal fluid was examined by Dr. G. M. Richards, of the Commonwealth Health Laboratory, and he reported that a great number of pus cells and numerous organisms (pneumococci) were present on the film, and that culture yielded a good growth of pneumococci.

At four-hourly intervals five cubic centimetres of 10% "Soluseptasine" solution were injected intramuscularly during the next twenty-four hours. Sixteen hours after operation he was given four tablets of "M & B 693", followed by three tablets four hours later, then two tablets every four hours. That day his temperature rose to 39·2° C. (102·6° F.), the pulse was more regular, headache was still severe and the patient was irrational at times; his bladder had to be catheterized. Lumbar puncture was again performed and 20 cubic centimetres of turbid fluid were obtained. Morphine was given for the headache and restlessness.

Next day (December 18, 1938), although headache was still very severe, the patient was more rational and passed urine normally. "Soluseptasine" and the tablets were continued every four hours.

On December 19, 1938, the patient's temperature remained normal all day, his headache was less severe, and he was quite rational. A lumbar puncture then showed the cerebro-spinal fluid to be much less turbid. On examination the fluid contained fewer pus cells and very few organisms. A very poor growth was obtained on

culture. "Soluseptasine" was again given intrathecally and the treatment was continued as before.

Two days later, when lumbar puncture was performed again, thirty cubic centimetres of clear fluid were obtained. On examination no pus cells or organisms could be found. Each day the patient said he was feeling better. Treatment was discontinued.

Although the patient's general condition was quite normal, his temperature again began to rise, so two tablets of "M & B 693" were given three times a day for the next three days. On December 29, 1938, his temperature was normal, and since then convalescence has been uninterrupted.

Comment.

I consider the points of interest in this case are: (*a*) the fact that the patient recovered from such an acute infection, which is rare, and (*b*) the rapidity with which signs of improvement were shown and the cerebro-spinal fluid became sterile.

Reviews.

GENITAL ABNORMALITIES.

The authoritative monograph on genital abnormalities, written by Professor Hugh Hampton Young, is a work which should be of great value to the physician, the surgeon, the gynæcologist and the urologist.[1] The subject is one of general interest not only to the medical profession, but also to our legal brethren and others, for, as the author has stated in the preface: "Intersexuals have intrigued the intellectuals since time immemorial; in fact quite a few intellectuals have been intersexuals."

After an introductory chapter dealing with hermaphroditism in literature and art, the embryology of the condition is detailed. Male and female pseudohermaphrodites and hermaphrodites of undetermined sex are then described. A chapter devoted to the discussion of reported cases of *hermaphroditismus verus* includes an account of the author's own case, the ninth accepted as proved microscopically. This case more clearly approximates to the perfect hermaphrodite than any other which has been reported and is one of the three cases of *hermaphroditismus verus lateralis*, a condition which is characterized by a single testicle on one side and a single ovary on the other. The patient resembled a male, but, owing to the suspicion that an undescended testis existed on the left side, it was decided to operate, and an ovary, tube and uterus were found. These organs were removed with good result.

In discussing the operative treatment for the adrenogenital syndrome, the author emphasizes the importance of an accurate knowledge of the condition of both adrenals and describes a technique for simultaneous bilateral exposure, study of and operation upon them.

Numerous interesting case reports are included in the text, for example, in the chapter on vaginal abnormalities in hermaphroditism, the case of a pseudohermaphrodite brought up as a female is described. At the age of eighteen years, as she was desirous of marrying a man, an enlarged "clitoris" was amputated at her request. At operation testes were discovered in the groins and their removal was insisted upon by the patient. A small vagina which was enlarged by operation was the only female organ present. Nevertheless, the patient's sexual appetite and habits were entirely female. Such cases as these give the reader food for thought as to the relation of the gonads to sexual function.

Separate chapters are devoted to masculinization due to ovarian tumour, prostates in females, hypergenitalism, hypogenitalism and gynæcomastia. The pathology and operative treatment of hypospadias, epispadias, cryptorchidism, exstrophy of the bladder and other congenital abnormalities of the genital tract are described in detail.

In the last chapter, which is devoted to the discussion of the relation of the genital tract to the endocrine glands and endocrine therapy, the author warns his readers that: "With the marked discrepancy between experimental endocrinology and hormone therapy, it is necessary that each clinical trial be based upon fundamental knowledge and careful observations rather than the haphazard, overenthusiastic reports that appear in the literature."

The text is profusely illustrated by 379 plates and 534 drawings and the work is a notable addition to the literature of a very important subject by a master.

GRAY'S ANATOMY.

While never a static text-book, "Gray's Anatomy" has manifested a rapid and progressive improvement in all its departments during the past ten years under the vigorous editorship of Professor T. B. Johnston.[1] There has been a continual replacement of the older figures by fresh and more valuable ones in successive editions; in the present about one hundred figures have been so introduced with great benefit, although more remains to be done, for example, the figure of the facial muscles is very obscure. For the first time a series of X ray plates has been included; this reform was overdue and could be extended. The section on the lymphatic system has been rewritten by Dr. Whillis, and the recent work of Le Gros Clark on the thalamus is included for the first time. It is interesting to observe that the short notes on "practical applications" appear to undergo progressive reduction in successive editions. The value of these in a text-book of this nature must remain a matter of personal opinion; but if they are necessary at all it would appear preferable to include them in a dissecting manual, so that the student could apply them on the material actually in hand, rather than in a text-book which already possesses a section on surgical anatomy. Surely it is the duty of the surgeon rather than of the anatomist to indicate what use a student is expected to make of his anatomical knowledge.

Anatomical teaching has been changing its viewpoint during the past few years; emphasis is laid more and more upon the body as a whole functioning organism rather than upon the minutiæ of detail demanded by earlier teachers. The subject has undergone a renaissance in outlook and method, a renaissance still in its infancy but already offering the prospect of a complete rejuvenation of this ancient science. The editors of "Gray" have always striven to keep abreast of contemporary advances and the new edition exhibits every determination to maintain this progressive attitude, especially in the provision of liberal references to sources of recent information. And it is pleasing to record that "Gray", apparently alone, promulgates the views of Allen *et alii* on the simultaneous development of several ova (of which only one normally attains maturation, however) in contrast to the generally accepted belief in the isolated monthly development of a single ovum only.

It would be easy to raise criticism on many minor points in a book of this size, but nothing can detract from its value as a whole. During the past eighty years it has fulfilled the requirements of many generations of students, and it gives every indication of performing this function for as many generations more. It is only necessary to add that the new student will find here practically everything he requires, while the older student, seeking fresh inspiration, will not look in vain.

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[1] "Genital Abnormalities, Hermaphroditism and Related Adrenal Diseases", by H. H. Young, M.A., M.D., Sc.D., F.R.C.S.I., D.S.M.; 1937. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 690, with 534 illustrations. Price: 45s. net.

[1] "Gray's Anatomy, Descriptive and Applied", edited by T. B. Johnston, M.D., assisted by J. Whillis, M.D., M.S.; Twenty-Seventh Edition; 1938. London: Longmans, Green and Company. Royal 8vo, pp. 1566, with 1336 illustrations, of which 624 are in colour. Price: 45s. net.

The Medical Journal of Australia

SATURDAY, MARCH 18, 1939.

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THE MENTAL HOSPITALS OF NEW SOUTH WALES.

NEARLY four years ago we were constrained to make violent protest in these pages about the shameful neglect by governmental authorities in New South Wales of the mental hospitals department. In none of the other States of the Commonwealth has the head of the mental hospitals department found it necessary to issue such a strongly worded report as that issued by Dr. C. A. Hogg in 1934. Never, we are certain, was a report from the medical head of a government department more justified in fact. In commenting on Dr. Hogg's devoted service in the public welfare, we remarked that he had bequeathed to his successor a legacy of blasted hopes. When Dr. John A. L. Wallace succeeded Dr. Hogg in the high office of Inspector-General he doubtless expected to see some result from the urgent plea of his predecessor. Probably this accounts for the restraint which has characterized most of his published utterances, for we cannot suppose that restraint was adopted at the command of a ministerial superior. But "hope deferred maketh the heart sick" and Dr. Wallace is sick at heart if we can judge by his report for the year ended June 30, 1938, which has recently been presented to Parliament.

Dr. Wallace's plaint is mainly of inadequate and antiquated accommodation; we recognize it as the same woeful tale that he has told before in part and that Dr. Hogg told before him. The number of new admissions of certified patients for the twelve months covered by the report is 137 greater than the number for the previous twelve months. In addition voluntary patients increased by 49 and inebriate patients by 43, the total increase for the period thus being 229. Dr. Wallace finds it difficult to associate the variations in the number of admissions with any dominating factor since conditions in the community do not vary from year to year. He adds that the alleged increase in insanity in other countries, when analysed, is misleading, and that there is no real increase. Mental defect and disease are recognized earlier and many persons who were formerly allowed to look after themselves are now cared for in mental hospitals. The most disturbing part of this report is the fact that permanent accommodation is provided in the department's institutions for 9,636 persons and that 10,961 are received, an excess of 1,325 patients. The variations in overcrowding of mental hospitals for the last twenty years is shown in a table. With the exception of the years 1928 to 1932, the years of the financial depression, the present excess is greater than it has ever been. In another table Dr. Wallace sets out the number of additional beds provided during the last five years. In the twelve months under review 92 new beds were supplied and none were provided by remodelling; the increased overcrowding for the year was 239. For more than twenty years the mental hospitals of New South Wales have been overcrowded and very little has been done to remedy matters. That the really shocking state of affairs may be realized it will be well to refer in detail to some of Dr. Wallace's statements—we had almost called them revelations, but they have been revealed so often that the use of the word in this connexion would be ridiculous. At the mental hospital at

Gladesville "the overcrowding of the female division remains in the same unsatisfactory state, being detrimental to chances of improvement or recovery of the patients". Dr. Wallace goes on to state that the accumulation in these old wards of large numbers of female patients, the majority of whom are in a condition of chronic mania, is a state of affairs which should be rectified at the earliest possible opportunity. "The chronic, excited and refractory type of patient must be allowed the maximum amount of space in dormitory, dayroom and airing court accommodation. It is found from experience that many of these cases improve remarkably in new surroundings where there is ample room to move about. The only solution of this difficulty is the commencement and completion of the proposed new female refractory ward, for which detailed plans have been ready for some considerable time." If this is not done the Government might consider the alteration of the name of the institution and call it the Gladesville Hostel for Mental Patients, instead of calling it a hospital. At the Parramatta hospital a considerable section of what are known as the old chronic female wards, housing altogether 456 patients, "is of weatherboard and in the event of fire would be a menace to the safety of the inmates". At Kenmore no progress has been made with the rebuilding in brick of the artisans' workshops which are constructed of weatherboard. "In the event of fire, these workshops would constitute a dangerous risk to the safety of the old men who are accommodated in the neighbouring weatherboard ward." It is, so Dr. Wallace states, "an urgent matter that this necessary work should be undertaken as soon as possible". Additional accommodation is urgently required for the segregation of male patients at Rydalmere. At Stockton the nurses' quarters are "totally inadequate" and one building had long been condemned as unfit for occupation.

The public is informed by *The Sydney Morning Herald* that on the day after Dr. Wallace's report was tabled in Parliament the Minister for Social Services and Acting Minister for Health, the Honourable Athol Richardson, announced that £45,000 was to be spent on extra accommodation for patients in mental hospitals which had been reported by Dr. Wallace to be overcrowded. The sum of £20,000 is to be spent at Stockton on two female nursery wards to accommodate 100 patients. Two "female refractory wards" are to be built at Gladesville, each with 50 beds, and these will cost £15,000. A female admission block for the accommodation of 60 patients will be erected at Callan Park Mental Hospital at a cost of £10,000. The sum of money promised by the Government will be little more than a drop in the ocean of mental hospital antiquity. The promise is a graceful *placebo*, a nice piece of political window dressing. The proposed new beds number 260, and these will do little more than cover the increased overcrowding of the last twelve months. The excess of patients for whom no provision is to be made is 1,065. But, it may be objected, the Government is starting to make good the deficiency. Of this we are not so certain. We have it from Dr. Wallace that detailed plans for the new refractory ward at Gladesville have been ready "for some considerable time". We also know that last year the Government promised to make available to the Mental Hospitals Department a sum of money much greater than that mentioned by Mr. Richardson, and we remember that only a fraction of the money then promised was forthcoming. If the Government were to provide 260 new beds every year for several years, and if it would pull down some of the relics of ancient days and substitute modern buildings in their places, the people of New South Wales would know that the mentally afflicted in the community were receiving proper treatment. It is clear that the medical officers of the department work under heavy handicaps. In spite of difficulties and in the face of official apathy, almost amounting to discouragement, attempts are made to introduce the latest methods of treatment and a great measure of success is achieved. Success would be greater, however, if the accommodation of the patients was adequate and if their surroundings were conducive to their recovery rather than a hindrance to it. As we have stated over and over again, it is the duty of the State to take more than usual care of mentally afflicted persons, who cannot care

for themselves, who cannot protest if they are neglected, and who cannot cast political votes of approval. The future will show whether the Government is sincere.

Current Comment.

PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY.

Pseudohypertrophic muscular dystrophy, or paralysis as the disorder was originally termed, is a myopathic disease whose pathogenesis is not yet understood. It would appear to be more common in boys than in girls, and may be manifested in several members of the one family and may appear in successive generations. An affected female is unlikely to bear children, but the disorder is transmitted through healthy females rather than through males.

L. Scheman, P. Lewin and S. Soskin have made a study of a group of five patients under controlled conditions.[1] They remark that the treatment of this dystrophy has passed through many phases in accordance with the prevailing idea as to its pathogenesis. For a time all the attention was focused on endocrinological factors and almost every gland was suspected. In particular the pineal and pituitary glands have been singled out by different workers. Other writers have emphasized a supposed disorder in carbohydrate metabolism. Deductions have been drawn from the occurrence of creatinuria in such unrelated conditions as diabetes and starvation. Occasional instances of this dystrophy have been recorded as following encephalitis and anterior poliomyelitis. Hence some observers have postulated a lesion in a hypothalamic centre. Some investigators have treated patients suffering from this disorder by adrenaline and pilocarpine, on the assumption that the disease originated in a lesion of the autonomic nervous system. Favourable results have been reported from this method of treatment, but the experience of Scheman and his colleagues has been disappointing. During the past few years encouraging reports have been made regarding the use of aminoacetic acid in the treatment of primary myopathies, especially *myasthenia gravis* and pseudohypertrophic muscular dystrophy. Scheman and his associates likewise reported favourable results. They noted, however, that, as their investigations proceeded, the progress was not maintained and that improvement was of only brief duration in the face of advancing atrophy and fibrosis. The interest of Scheman, Lewin and Soskin was revived by the investigations of G. Meldolesi in Italy dealing with over 100 cases spread over nine years. Meldolesi considered that the muscular dystrophies were the result of a primary disorder of the pancreas, whereby tryptic and lipolytic digestive processes were conspicuously impaired. Failure of adequate protein absorption causes a depletion of protein reserves with a resulting dysplasia of striated muscle. This occurs in persons with an hereditary and familial diathesis of which the basis is a lack of pigment of the muscles. Meldolesi noted that the curves for the secretion of lipase, diastase and trypsin were delayed in their rise and failed to reach normal levels. Many of the patients exhibited diminished dextrose tolerance. The fæces contained an increased quantity of neutral fats and fatty acids. In the urine there was a diminished total excretion of nitrogen. With the initiation of therapeutic measures creatinuria rapidly lessened and within a few weeks only a negligible amount was excreted. Treatment was intended to correct the deficient digestion of protein and the altered carbohydrate metabolism. The treatment adopted was the exhibition of 40 to 60 drops of pepsin-pancreatin twice daily, with insulin and dextrose given by the intravenous route.

Scheman and his associates made an extensive investigation of the group of five patients mentioned in the light of Meldolesi's contentions. They conclude that patients suffering from pseudohypertrophic muscular dystrophy do not lack the ability to digest proteins or to transform these proteins into the end products of ferment activity. They further state that there is apparently no disturbance in the formation of creatine. This was readily observed from the definite and proportionate manner in which creatinuria followed nitrogenous intake. It had long been considered that both creatine and creatinine originated solely in endogenous metabolism; but the observations of Scheman and his colleagues, as well as those of other investigators, would indicate that there must also be some exogenous source. When creatine and creatinine are expressed in terms of the percentage of total nitrogen excreted they bear an inverse relationship to the latter. Scheman and his colleagues further state that diminution in the intake of protein causes a fall in total excretion of nitrogen and in that fraction of the latter which represents exogenous creatine and creatinine. Endogenous katabolism remaining the same, the excretion of creatine and creatinine derived from it makes up a disproportionate fraction of the total excretion of nitrogen and hence bears an inverse relationship to the latter in terms of percentage. This is well seen when the protein-sparing action of large amounts of carbohydrate is involved. It has long been considered that in this disorder there is a disturbance of glycogenesis, and accordingly administration of carbohydrates, with or without insulin, has been strongly advocated. It is suggested that the conspicuous manner in which administration of carbohydrates stops the creatinuria of starvation is corroborative evidence. But Scheman and his colleagues can find no support for such theories. Nor did they note any correlation of clinical improvement with periods of high carbohydrate ingestion. They further state

[1] *The Journal of the American Medical Association*, December 17, 1938.

that, in their experience, no form of therapy available at the present time acts in a specific way to halt the progress of the disease. Regulated exercise is important, as is abundantly evident when one notes the harmful results of restricted activity on biopsy of the gastrocnemius muscle. Scheman and his colleagues particularly mention a number of tissue pigments and especially a muscle pigment (myoglobin) which is endowed with a respiratory function. They insist that when more is known of the place occupied by such pigment substances in the economy of muscular processes an important clue will have been found for the treatment of myodystrophic patients. We should like to think that we are within measurable distance of elucidating the pathogenesis of this crippling disorder.

THE SERUM TREATMENT OF HÆMOLYTIC STREPTOCOCCAL INFECTIONS.

Since the introduction of sulphanilamide comparatively little importance has been attached to the treatment of hæmolytic streptococcal infections with specific antisera. There are, however, some persons who cannot tolerate sulphanilamide because of idiosyncrasies, and in some rare instances of hæmolytic streptococcal infection sulphanilamide, though well tolerated, is ineffective. Reuben Ottenberg[1] believes that in every severe hæmolytic streptococcal infection it is wise to supplement chemotherapy with serum treatment. He observes that there is some evidence that the drug and serum treatment supplement each other; the serum is antitoxic, while the drug has a bacteriostatic effect. Certainly the two forms of treatment should not interfere with each other. Ottenberg recommends the use of convalescent scarlet fever serum when this is available. He states that the trend of recent clinical opinion is that scarlet fever convalescent serum is superior (despite its lower antibody content) to antitoxin prepared from the serum of animals. Most observers agree that ordinary polyvalent antistreptococcus serum has too little value to merit serious consideration. The number and variety of the strains of hæmolytic streptococci found in human infections offer at least a partial explanation of the ineffectiveness of most polyvalent antistreptococcus sera. The experience of recent years with type-specific antipneumococcus serum shows how unlikely it is that any polyvalent antibacterial serum will contain enough antibody to be of more than accidental value in the treatment of infections caused by organisms of such widely varying serological strains as the hæmolytic streptococci.

It is difficult to understand why the serum of patients who are convalescent from scarlet fever should be of more value than polyvalent antistreptococcus serum derived from laboratory animals; yet there seems to be some clinical evidence that this is so. It is possible that this evidence is based on the treatment of severe hæmolytic streptococcal infections forming part of the same epidemic as the cases of scarlet fever from which the antiserum was derived. Such serum would probably be type-specific for the prevailing epidemic strains of hæmolytic streptococci.

Early bacteriological diagnosis and treatment with sulphanilamide will prevent the occurrence of severe symptoms in hæmolytic streptococcal infections. Sulphanilamide, however, has not proved to be very effective in the treatment of scarlet fever. On the whole, it would be a wise precaution, at the commencement of an epidemic of scarlet fever, to collect serum from convalescent patients and to hold it in readiness for the treatment of severely ill scarlet fever patients and for subsidiary treatment in any severe hæmolytic streptococcal infections that might occur, in spite of all precautions, during such an epidemic.

[1] *Bulletin of the New York Academy of Medicine*, August, 1938.

TROPICAL MACROCYTIC ANÆMIA.

One of the difficulties in the ætiology of anæmia is that anæmia, whatever its type, is in most instances merely a manifestation of disease and not a disease in itself. In reading recent literature on anæmia one gains the impression that this simple fact is often overlooked by modern investigators. There are certain anæmias which, in the light of our existing knowledge, have to be considered as diseases; but it is quite possible that even they, as more is learnt about them, will come to be regarded rather as symptoms. Tropical macrocytic anæmia is a disorder that is well known to medical practitioners in tropical countries. The general opinion has been that it is caused by a deficiency of Castle's extrinsic factor. This is an agreeable hypothesis, but not necessarily a true one. Consideration must be given to the possibility that macrocytic anæmia (apart from pernicious anæmia) in the tropics might have numerous types of origin. L. Everard Napier suggested in 1936 that tropical macrocytic anæmia might be due to a deficiency in an independent hæmatopoietic principle. After further investigations he has formed the conclusion, amongst others, that "there is a close relationship between the hæmolytic and non-hæmolytic types of tropical macrocytic anæmia and that malaria is an important predisposing factor in the former".[1] This view is suggestive of a tendency towards a belief in a multiplicity of causes.

Napier points out that if current views are accepted, the pathological changes caused by absence of the hæmatopoietic principle should be identical, whether the deficiency is of the intrinsic or the extrinsic factor, and that any constant differences between tropical macrocytic anæmia and pernicious anæmia must be attributable to the

[1] *The Indian Medical Gazette*, January, 1939.

gastric changes in pernicious anæmia. He then proceeds to discuss various points of similarity and differences between these two conditions. Both have the features common to all other types of anæmia. The so-called "raw beef" tongue is occasionally seen in tropical macrocytic anæmia, and the oral or parenteral administration of crude liver extract is an effective therapeutic measure in both. The outstanding point of difference in the symptomatology is the absence of any neurological disturbance in the tropical anæmia. There are numerous differences in the blood picture. The mean red cell volume is high in both conditions, although the mean cell diameter in tropical macrocytic anæmia is only slightly above normal; "that is to say there is a spherocytosis in tropical macrocytic anæmia". Poikilocytosis and anisocytosis are less pronounced in the anæmia of the tropical type. The incidence of achlorhydria in tropical macrocytic anæmia is the same as in the general population; but Napier found a slight diminution of the gastric acid secretion as a rule. Yeast extract or "Marmite" alone will cure a patient of tropical macrocytic anæmia; massive doses will sometimes evoke a response in pernicious anæmia. Napier points out that while it is admitted that Castle's extrinsic factor is contained in "Marmite", it is possible that there is another factor, which evokes hæmatopoiesis in tropical macrocytic anæmia. "Anahæmin" and comparable substances have little or no effect in tropical macrocytic anæmia, whereas ordinary liver extract has. The suggestion is that both factors are present in liver, but that the one concerned in tropical macrocytic anæmia is absent from "Anahæmin" and like preparations.

In uncomplicated tropical macrocytic anæmia there is usually no great excess of bilirubin in the blood, whereas in pernicious anæmia bilirubin is present in comparatively large amounts and the Van den Bergh reaction is pronounced. This hyperbilirubinæmia is said to be due to failure of the hæmatopoietic system to utilize the products of normal hæmolysis. Napier has evolved a more attractive theory. He suggests that there is excessive hæmolysis, "that the defective products of hæmopoiesis are readily destroyed by a relatively normal reticulo-endothelial system". He points out that if his theory is correct there is a ready explanation for the lower concentration of bilirubin in the blood in tropical macrocytic anæmia; for here, though the cells are larger than normal, anisocytosis and poikilocytosis are less pronounced, and there are "fewer defective cells to attract the attention of the normal reticulo-endothelial tissues".

Napier then proceeds to discuss what he refers to as the hæmolytic group of tropical macrocytic anæmias. He postulates the existence of a "substance (or group of substances) normally present in marmite and crude liver extract which is required *quantitatively* for the normal maturation of red cells at the megalo-erythroblast level". He suggests that this substance may be stored in the body, and he goes on to say:

> A normal mixed diet contains sufficient of this substance for normal requirements, and, together with the possible storage reserve, for emergencies such as hæmorrhages and moderate hæmolyses, but in severe and chronic hæmolyses—which demand a much greater corpuscular replacement than any non-fatal hæmorrhage—the reserve is rapidly exhausted and there may be a deficiency, which will be exaggerated if there is a deficiency of this substance in the food. A diet grossly deficient in this substance will also in time give rise to pathological changes, which will be exaggerated by conditions of extra demand such as pregnancy, or of mal-absorption, as in sprue and lienteric diarrhœa.

Napier suggests as future methods of research into the causes of these anæmias a continuance of the investigation of the nutritional anæmia of monkeys along the lines of Wills's experiments, and clinical experimental work with purified fractions of liver extract and autolysed yeast used quantitatively. His opinions are of value and they will no doubt assist in the elucidation of the ætiology of the anæmias. The need for some means of elucidation is apparent in this as in most papers on anæmia. There is obvious confusion, and this has partly resulted from a persistence in the consideration of all forms of anæmia as disease.

A MAGNIFICENT BEQUEST.

After living in retirement for more than thirty years Dr. James O'Neil Mayne died recently in Brisbane at the ripe age of seventy-eight years. He will be remembered for all time as one of the great benefactors of Australian universities. He first showed his generosity many years ago when he paid for the first X ray plant that was installed at the Brisbane General Hospital. He was resident medical officer and superintendent of that institution, and during the whole of his term of office gave the whole of his salary to be spent in improvements to its buildings and grounds. It was he who gave to the University of Queensland the magnificent site at St. Lucia on which buildings are being erected. He also gave to the University of Queensland 693 acres of land to be used for agricultural purposes. On his death he left his entire estate, apart from personal effects, to the medical school of the university. According to the Brisbane *Courier-Mail* the estate is valued at upwards of £100,000. The estate will be administered by trustees and will be devoted particularly to the purchase of equipment for use in the medical school, to the establishment and maintenance of chairs of medicine and surgery, to the endowment of medical research, and to the foundation of scholarships within the medical school.

The people of Queensland and of Australia have reason to be grateful to James O'Neil Mayne, who, living among them in quiet and unobtrusive fashion, showed his love of country by trying to make provision for the future welfare of its citizens. By wise administration and devotion to the ideals that actuated him they "may counterpoise this rare and precious gift"; and perchance other of his colleagues may follow his generous example.

Abstracts from Current Medical Literature.

SURGERY.

Tube Pedicle Skin Grafts.

Thomas D. Sparrow (*The American Journal of Surgery*, July, 1938) pays tribute to M. Filatoff and Howard Gillies for the introduction of the tube pedicle graft. Gillies claims many advantages for this mode of skin grafting. Large quantities of skin may be transplanted. The tube lends itself to temporary attachment through an "intermediate host", such as the arm, and thus can be transported safely a great distance. It is particularly useful where no massive direct flaps are available. Its flexibility allows it to be twisted and turned in the most liberal manner without causing anxiety as to its viability. The author points out another advantage that has not been sufficiently stressed. He reports a case in order to illustrate and emphasize the extraordinary ability of the tube pedicle graft to grow in the most unfavourable surroundings and in an environment that would preclude the use of any other grafting method. The case reported was that of a woman who had had a complete excision of the skin about the vulva and rectum for *kraurosis vulvæ*. The resultant scarring was painful and frequently became ulcerated. Two crescentic pedicle flaps were formed from the healthy skin of her buttocks and four weeks later transplanted to the posterior edge of the rectum. After three weeks the scar tissue was removed and replaced by the unfolded tube. Healing was complete within six weeks, although during this time the transplanted skin was constantly liable to infection by contact with fæces.

Back Strain.

Joseph S. Barr (*Surgery*, July, 1938) describes intractable low-back and sciatic pain that is occasionally due to the mechanical pressure of intervertebral disk tissue protruding posteriorly into the spinal canal and impinging on one or more roots of the *cauda equina*. The symptoms and signs of a ruptured intervertebral disk are so constant and characteristic that the presumptive diagnosis can often be made before lipiodol studies are made. The typical patient is a vigorous man in his thirties, who has been perfectly well until, while lifting a heavy weight, he feels something snap in the lower part of his back and has immediate pain in the lumbo-sacral region. After an interval unilateral sciatica develops, the pain commencing deep in the buttock, radiating down the back of the thigh and occasionally to the postero-lateral aspect of the calf and the lateral border of the foot. The pain is accentuated by certain movements. It may come in attacks, and generally fails to respond to the usual therapy. On examination the patient is found to stand with his trunk thrust forward and to one side, with most of his weight borne on the opposite leg. The normal lumbar lordosis has been lost, and in its place is a fixed reversal of the lumbar curve with great prominence of the spinous processes of the third, fourth and fifth lumbar vertebræ. There is also a little tenderness at the sacro-sciatic notch on the affected side. Inspection reveals some atrophy of the buttock, thigh and calf of the painful side. The ankle jerk is absent on the painful side and normal on the other. The other reflexes are all normal. No sensory or motor changes are found. Straight leg raising can be carried through an arc of 70° on the non-painful side, and through 20° to 30° on the painful side. An attempt to force the leg upward beyond this point causes a sharp increase in the sciatic pain. The usual antero-posterior and lateral Röntgenograms of the lumbar part of the spine and the pelvis show no changes from the normal, except an abnormally flat appearance of the lumbar part of the spine in the lateral view and possibly slight narrowing of one or both of the two lower intervertebral spaces. A history and physical examination similar to this suggest strongly that the diagnosis is posterior rupture of one of the lower lumbar intervertebral disks. Lumbar puncture and lipiodol studies are then made to confirm the diagnosis. Treatment is by laminectomy with removal of the protruding tissue. In a series of some 58 proved cases at the Massachusetts General Hospital, 54 patients either recovered or were greatly improved. Only three patients were unimproved, and there was one operative fatality.

The Spleen and Shock from Hæmorrhage.

E. P. Lehman and C. V. Amole (*Surgery*, July, 1938) present the records of an experimental study of the effect of hæmorrhage in dogs from which the spleen had been removed. It is known that within a few months the immediate post-splenectomy qualitative changes in the blood, such as anæmia and increased resistance of the red cells to hæmolysis, disappear and the blood becomes practically normal. It is believed that other portions of the reticulo-endothelial system take over these functions. These other tissues cannot, however, assume the reservoir function of the spleen. By reason of its capacity to change its volume and its by-path position in the circulation, the spleen is able to store blood and to deliver it promptly to the circulation when there is need. So far as is known this function remains in default after splenectomy. The experiments indicate that dogs whose spleens have been removed sustain blood loss by repeated withdrawals less well than the intact dog, although the reservation is made that it would be desirable to have the results supplemented by studies at a longer interval after splenectomy to confirm the accepted opinion that later compensations for the lost reservoir function are not developed. It is suggested that the individual who has lost his spleen should be looked upon by the surgeon as possibly a somewhat poorer risk for operation or accidental trauma on that account, and that the surgeon should be on the alert for an earlier development of shock than he would otherwise expect.

Exclusion of Pancreatic Secretion.

F. F. Boyce and E. M. McFetridge (*Surgery*, July, 1938) excluded the pancreatic secretion from the intestines of a number of laboratory animals to study the effect on protein and fat metabolism. The outstanding considerations in various types of cancer of the ampulla and peri-ampullar regions are outlined, with particular reference to the physiological disturbances that follow the necessary radical surgery for the removal of these tumours. The types of operation devised for these lesions are considered from the standpoint of the immediate surgical mortality. In all the animals studied the digestion of fat and protein was approximately normal, even when a diet unusually rich in these substances was given. In spite of this fact, fatty infiltration occurred in the liver of animals in which partial or complete pancreatectomy was performed; but the addition of lecithin to the diet of these animals was found to prevent such a change. No changes occurred in the metabolism or in the liver of animals in which the pancreatic ducts were ligated and divided, the pancreas being left *in situ*. This evidence is in keeping with Dragstedt's assumption of a pancreatic hormone that is able to take over the function hitherto assigned exclusively to the external secretion. It is suggested that radical surgery that does not involve excision of the pancreatic tissue may safely be performed without reimplantation of the pancreatic ducts for malignant disease of the ampullar and periampullar regions. In such cases fatty infiltration of the liver is not likely to develop. It is also suggested that when such surgery must include excision of the head or of the head and body of the pancreas, fatty changes will develop in the liver unless lecithin, choline or pancreatic substance or alcoholic extracts of pancreas are administered to forestall them.

The Correction of Saddle Nose by Ivory Implant.

Richard M. Wolfe (*Archives of Surgery*, November, 1938) believes that pure elephant ivory offers the ideal implant for the correction of such deformities as saddle nose. Autoplastic materials, such as bone and cartilage, often produce an abnormal mid-line ridge, due sometimes to the narrowness of the graft, but most frequently to absorption. The author describes a one-stage operation in

which an ivory implant is used. He states that ivory has been used for twenty-five years and has stood the test of time, and that it retains its original shape and size. It is essential that genuine elephant ivory be used, preferably from young elephants; and the author indicates a test in which sulphuric acid is used to determine that the ivory is genuine. He makes an impression of the patient's face, and then, using artificial stone, takes a cast. Having selected a piece of ivory, he smears the dorsum of the nose of the cast with lipstick. The ivory is placed on the nose, and where the lipstick leaves an imprint the ivory is carved. For this he uses a rotary motor with carborundum wheels. After repeated fitting and carving the under-surface, when completed, will fit the saddle depression with the accuracy of a dental inlay. The superior surface is then carved to a height and width equal to the depth and width of the deformity and is then drilled with tiny holes to make it lighter. Two implants are made in every case, one an accurate fit and another slightly oversized. The implants are scrubbed and then smoothed down with a sandpaper disk, a dental engine being employed. The ivory implants are sterilized by slow boiling, and in the preparation of the patient the hairs in the nostrils are clipped and the nose is packed for half an hour with 1 in 8,000 mercurophene solution. On the operating table the part is cleaned with alcohol, "Metaphen" (1 in 200) and again with alcohol. General anæsthesia is not used, as post-operative nausea and vomiting invite infection. A 1% solution of "Procaine" with epinephrine is administered intranasally to the sides and dorsum of the nose and an incision is made inside the left nostril at the inferior border of the triangular cartilage. Blunt dissection permits the introduction of the implant, and the incision is not sutured.

Extradural Hæmorrhage.

K. G. McKenzie (*The British Journal of Surgery*, October, 1938) gives short histories of patients suffering from extradural hæmorrhage, and comments on them. Of the patients, nine died; three died owing to failure of the surgeon to operate, and three owing to delay in operation; two deaths were due to concomitant severe injury, and one was due to extension of the clot to the cerebellum. The differential diagnosis of this condition includes bruising and œdema of the brain, intracerebral hæmorrhage and subdural hæmorrhage. A lucid interval after injury is the most valuable diagnostic indication, though it may occasionally be absent. A dilated pupil is a very valuable lateralizing sign. Paresis may be useful for diagnosis; it is difficult to establish in a stuporous patient, and it may be a false localizing sign; it may be on the same side as the lesion, in which case it is usually caused by the *contrecoup* bruising of the hemisphere. Clear or faintly blood-tinged cerebro-spinal fluid in the presence of a lucid interval followed by stupor makes the diagnosis almost certain; on the other hand, if the fluid is very bloody the diagnosis is in doubt. A fracture as revealed by X rays is a valuable sign, both diagnostic and lateralizing, though cases occur without fracture. The mortality rate indicates the seriousness of the lesion and illustrates the necessity for accurate and early diagnosis and prompt treatment. Bilateral exploratory burr holes should be made if there is a reasonable suspicion of the diagnosis.

Non-Operative Treatment of Perforated Gastric Ulcer.

Patric Nagle (*Surgery*, November, 1938) reports the history of a man who suffered from an acute perforation of a gastric ulcer into the free peritoneal cavity and who was successfully treated without operation forty hours after perforation by continuous suction applied to an indwelling duodenal tube. While insisting on the early closure of all perforations of the gastro-intestinal canal by open operation, the author points out that leakage from the closure is aided materially by the maintenance of continuous evacuation of the stomach and duodenum. In cases that seem too late for the surgical closure of the perforation the prognosis is materially improved by maintaining a continuously empty stomach and duodenum. The technique followed is that of the Wangensteen nasal suction siphonage.

Osteopsathyrosis.

Karl M. Lippert (*Surgery*, November, 1938) presents full reports of three cases of osteopsathyrosis or *fragilitas ossium*. He describes three groups. Patients in the first or congenital group are born dead or die soon after birth and are found to have received numerous fractures during delivery. Those of the second or infantile group are apparently normal at birth, but in their first few years experience many fractures and dislocations. Members of the third or juvenile group do not show the skeletal abnormality until late childhood or about the age of puberty. The disease is apparently inherited, and the author describes the condition of patients aged six years, nineteen years and forty-two years. He includes a genealogical table covering four generations, but states that the manner of genetic transmission is not evident. The X ray and microscopic pictures of the bones are relatively constant and unlike those of any other pathological entity. There is no biochemical evidence to show that the disease is related to errors of calcium and phosphorus absorption and retention. Blue sclera appear frequently, though not constantly, in the disease, and a few patients are deaf. The treatment of fractures in osteopsathyrotics is the same as that of fractures in normal individuals, and rapid healing is the rule.

Madelung's Deformity.

Joseph I. Anton, George B. Reitz and Milton B. Spiegel (*Annals of Surgery*, September, 1938) give a full description of Madelung's deformity and include a tabulation of 171 case reports. In reviewing the literature (and they give 239 references), the authors found it to be inaccurate, incomplete and replete with reports of cases that were not genuine. They describe the condition as an idiopathic progressive curvature of the radius due to a dyschondroplasia of the inferior radial epiphysis, resulting in a deformity of the wrist and giving it the appearance of an anterior, or more rarely a posterior, subluxation of the hand. They present a typical example of Madelung's deformity occurring in a girl of eighteen years, and they advocate the substitution of the term "dyschondroplasia of the distal radial epiphysis" in place of "Madelung's deformity", since the latter term is not specific. They suggest that this deformity should be placed among the group of conditions which includes Legg-Perthes's, Osgood-Schlater's, Köhler's and Keinboch's diseases.

Congenital Diaphragmatic Hernia.

Edward J. Donovan (*Annals of Surgery*, September, 1938) reports ten cases of congenital diaphragmatic hernia occurring in infants and young children. Six of these patients were operated upon; two died. The author states that most cases of diaphragmatic hernia are symptom-free and are discovered in the course of routine radiographic examination. If symptoms are present, they may be either respiratory or gastro-intestinal in origin, depending upon what viscera are contained in the hernia and what structures in the chest are pressed upon by the hernial contents. He considers that the treatment should be surgical repair of the hernia whenever any portion of the intestinal tract is involved, because of the danger of intestinal obstruction. He prefers the abdominal approach and positive pressure anæsthesia. All but one of the patients on whom the author operated were under six months of age, and he comments on the importance of the early performance of the operation for two reasons. In the first place intestinal obstruction may occur, and secondly, if the abdominal contents are allowed to remain in the chest for a long period, the development of the abdomen will be so retarded that it may not be large enough to accommodate the structures brought down into it from the chest. Of the ten patients, five manifested no abnormalities other than the diaphragmatic hernia. The others had further abnormalities such as mongolian idiocy, congenital heart disease, cleft palate, pilonidal sinus, hypospadias and hypertelorism.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on October 12, 1938, DR. COLIN MACDONALD, the President, in the chair.

Localized Arachnoiditis of Otitic Origin.

DR. MOSTYN POWELL showed a female patient, aged ten years, who had been admitted to the Children's Hospital originally on March 17, 1938. At that time she had had severe bilateral basal pneumonia followed by right basal empyema, and the empyema cavity had been drained in the fourth week of her illness. On August 21 she was readmitted to hospital, suffering from right-sided earache of six days' duration, severe frontal headache and pain in the neck of five days' duration, and persistent vomiting of one day's duration. Having spent three months at a convalescent hospital she had been at home only for two weeks.

On August 21 her temperature was 37·8° C. (100° F.), the pulse rate was 96 per minute, and the respirations numbered twenty per minute. She was apathetic and looked ill. The throat was injected and the right ear drum was redder in appearance than the left one. Apart from slight dulness and diminished vesicular murmur around the old rib resection scar, no abnormality was found on examination of the chest and abdomen. There was no neck stiffness, and Kernig's sign was absent. There was no weakness of the limbs; the deep reflexes and the superficial abdominal reflexes were equal and active, and the plantar responses were flexor. Sensation and coordination were apparently normal. Nystagmus was present; the coarse phase was apparent when the patient looked to the right, and consisted of a quick outward movement and a slow return. When she looked to the opposite side the nystagmus was fine and rapid. The visual fields were normal in extent; but some venous engorgement was noted on examination of the optic fundi. A provisional diagnosis was made of right *otitis media*, possibly associated with mastoiditis and a cerebellar lesion on the side of the coarse phase of the nystagmus. The cerebro-spinal fluid obtained by lumbar puncture was found to contain 108 leucocytes to the cubic millimetre, of which 58% were polymorphonuclear. Globulin was present in excess, and organisms, probably streptococci, were grown on culture.

Paracentesis of the right ear drum was carried out without delay and a little thin pus was obtained. For the following eighteen days the temperature was of the swinging type, the maximum being 38·7° C. (101·6° F.). The pulse rate varied between 80 and 100 beats per minute. The ear infection disappeared after four or five days, but the headache and vomiting persisted.

A series of skiagrams was prepared. It was found that the appearances in those of the chest were normal; some sclerosis in the right mastoid region and the presence of a congenital defect of the parietal bone were the only abnormal appearances in those of the skull. For one week the patient was given two tablets of sulphonamide every six hours. That treatment was then discontinued; but as she seemed to relapse it was recommenced a week later and continued until the temperature had fallen. Lumbar puncture was performed at intervals of four or five days, and on each occasion clear fluid was obtained under increased pressure. The number of cells gradually decreased; but the proportion of lymphocytes increased till they comprised 80% of the leucocytes. The manometric pressure of the cerebro-spinal fluid varied between 250 and 180 millimetres of water; but one week before the meeting the reading was 300 millimetres, so that it was apparent that increased pressure was still a feature of the case. Though the cocci obtained at first were probably streptococci, subsequently staphylococci only were grown on culture.

On the seventeenth day of the patient's stay in hospital, when great clinical improvement had occurred, papilloedema amounting to four diopters developed in the right eye, and to a slightly less extent in the left eye. The papilloedema continued, though it decreased considerably. From the eighteenth day after her admission to hospital the patient had no further elevation in temperature, discharge from the ear, vomiting or headache. Nystagmus had almost disappeared, and she could walk without unsteadiness. The visual acuity was $^6/_6$ on each side, and she was in apparent good health. The only remaining pathological findings were persistence of increased pressure of the cerebro-spinal fluid, blurring of the optic disks and retinal hæmorrhages, and very slight nystagmoid movements of the eyeballs.

Dr. Powell said that the patient's type of case was seen much more frequently at a specialist hospital, such as the Eye and Ear Hospital, than elsewhere. Discussion of such cases involved consideration of the complications of middle-ear or mastoid disease. Those complications formed a fairly well defined and limited group, though the differential diagnosis might be far from easy. For practical purposes they could be enumerated under ten headings: (i) incomplete removal of mastoid cells; (ii) extradural abscess; (iii) lateral sinus thrombosis; (iv) meningitis—(*a*) localized meningitis, which might progress no further, or which might be merely a stage in the formation of a cerebral or cerebellar abscess, according to the aspect of the petrous temporal bone on which the extension of the infection appeared, or (*b*) generalized meningitis; (v) subdural abscess, which rarely occurred alone; (vi) intracerebral abscess, either of the temporal lobe or of the cerebellum (hopefully subacute or hopelessly fulminating); (vii) non-suppurative encephalitis, the *abcès sans abcès* of Borries, the "blind boil" of the brain, indistinguishable from a true abscess except when proved by exploration, always affecting the temporal lobe and running a favourable course; (viii) otitic hydrocephalus of Simons, accompanied by characteristically increased intracranial pressure, and requiring treatment by repeated performance of lumbar puncture; (ix) petrositis, frankly osteomyelitis; and (x) simple septicæmia.

By piecing together the clinical course of the child's condition Dr. Powell had come to the conclusion that the likely train of events was the following. First, an acute *otitis media* of streptococcal or staphylococcal origin occurred; this was a common complication of pneumonia and empyema. Next, an extension took place, possibly via the bone between the labyrinth and the lateral sinus to the extradural region on the posterior aspect of the petrous temporal bone. Thirdly, a localized adhesive meningo-arachnoiditis supervened; this gave the original cerebro-spinal fluid findings and culture of coccal organisms. At that stage there were right cerebellar signs. The patient must have been on the point of developing a cerebellar abscess; but her resistance, aided by the sulphonamide therapy, had apparently been sufficient to cope with the infection. At that time she had been watched closely for the three indications for surgical exploration: (i) normal or very slightly raised temperature, (ii) increasing drowsiness, and (iii) a fall in the polymorphonuclear leucocytes and a rise in the lymphocytes in the cerebro-spinal fluid. Those indications had not appeared. Fourthly, at the end of the third week in hospital, when she was improving substantially, papilloedema had appeared, though she had not complained of headache and had not vomited. That happening might represent basal adhesions causing some degree of hydrocephalus. It was not impossible even at the time of the meeting that a localized abscess was present; but that was unlikely, as the patient had made such obvious progress, and the cytology and globulin content of the cerebro-spinal fluid had returned to normal. She conformed at that stage quite well to the picture of otitic hydrocephalus.

Dr. Powell said that the prognosis was good. He pointed out that the swelling of the optic disks was subsiding, and that though they might later appear pale, the visual acuity would probably remain unimpaired. He believed that if the skull could be examined a tiny patch would

be found on the posterior petrous temporal bone, in which adherent *dura mater* and arachnoid mater would be the evidence of an almost fatal illness.

DR. COLIN MACDONALD confirmed Dr. Powell's statement that the anomalies in the skiagrams of the skull were due to the presence of a congenital parietal foramen. He said that this was very uncommon.

DR. J. W. GRIEVE congratulated Dr. Powell on the way in which he had presented the patient. He said that it was not uncommon to have in the wards patients with diseases of the ear complicated by abnormalities in the cerebro-spinal fluid. It was often difficult to arrive at definite conclusions. A special point of interest in Dr. Powell's case was the lateness of the changes in the eye ground. The changes could easily have been missed if ophthalmoscopic examinations had not been persisted with.

DR. H. J. SINN said that the terms "serous meningitis" and "*ependymitis serosa*" might be applicable in such a case, though he preferred "otitic hydrocephalus" on account of the age and condition of the patient, the cerebro-spinal fluid findings, the degree of papillœdema and the absence of localizing signs. He agreed that the prognosis should not be gloomy, and that lumbar puncture should be performed repeatedly while headache and papillœdema persisted. If that failed to give relief, even ventricular puncture might be needed. In one such case pus had been obtained from an abscess at a later stage. If an increase in the cells or the protein content of the cerebro-spinal fluid took place, the diagnosis should be changed to one of cerebral abscess.

DR. PAUL JONES referred to an example of parietal foramen in the museum of the Royal College of Surgeons. The foramen was a large one, and the death of the man was said to have been due to a thrust from an umbrella which had been poked through it.

Dr. Powell, in reply, said that it was not wise to be too pedantic about the terms "otitic hydrocephalus" and "localized arachnoiditis"; broadmindedness was necessary. At one stage the patient had not a syndrome filling the requirements of the term "otitic hydrocephalus". The patient's resistance and the treatment might have arrested the formation of a cerebral or cerebellar abscess; but this could still occur.

Thymic Asthma.

DR. W. MCKILLOP showed a male baby who, in his opinion, was suffering from thymic asthma. He said that the baby had been born on March 24, 1938, and that the confinement was the mother's second and was normal. The baby was breast-fed and had made satisfactory progress until, on July 1, febrile respiratory catarrh had developed. Immediately afterwards he had the first of a series of attacks in which his body stiffened and he became "mucousy" and wheezed. He had a particularly severe attack on August 8; while he was awake his breathing was audible all over the room. There was no cyanosis and he could cry normally and sleep quietly. At that stage Dr. McKillop had consulted with Dr. Raymond Hennessy and Dr. Colin Macdonald had prepared skiagrams in which tortuosity and constriction of the treachea and widening of the opaque area in the upper part of the chest were apparent. A little later the baby had collapsed and appeared to be dead. When Dr. McKillop had reached him he had presented the typical picture of *asphyxia pallida*. His breathing was inaudible. Dr. McKillop had managed to hook the baby's palate forward and to introduce a catheter into the trachea. After a bath the baby's condition had improved considerably.

On August 19 a course of deep X ray therapy was commenced. This was continued until September 2. The baby also received before his meals a mixture containing 0·7 milligramme (one two-hundredth of a grain) of atropine sulphate in each dose. While he received that treatment the spasms had become fewer and had occurred with a greater interval of time between them; the baby had been taking the feedings better, but still usually had asthmatic wheezing.

Dr. McKillop thought that the baby had enlargement of the thymus gland, possibly of the thymoma type. Dr. Mollison had directed his attention to the fact that there was at times a syphilitic element leading to fibrosis of the gland; but the sera of the baby and mother had failed to yield the Wassermann reaction. According to the text-books, enlargement of the thymus gland associated with ganeralized lymphatism usually occurred in large, fat, flabby babies; but the baby he had shown that evening could not be so described.

DR. COLIN MACDONALD said that the diagnosis by X rays of an enlarged thymus as the cause of certain respiratory symptoms appeared to him to be fraught with considerable uncertainties and difficulties. These difficulties required to be mentioned, because in the living patient it was necessary to rely chiefly on the skiagram in the making of the diagnosis. The uncertainties were such that Dr. Macdonald made the radiological diagnosis of thymic enlargement with much the same reluctance as he did that of primary chronic appendicitis. Perhaps he was excessively shy of the thymus; but in sixteen years of radiology he had experienced the time when thymic enlargement had been diagnosed much too frequently by X rays. He had seen interest in the thymus wax and wane. It had, he felt, a certain periodicity; maximum interest usually followed the report of a so-called thymic death.

The clinician and the radiologist had to ask themselves three questions concerning this matter: (i) Was the broadening of the upper portion of the mediastinal shadow abnormal? (ii) If it was abnormal, was it due to an enlarged thymus? (iii) If an enlarged thymus was actually present, was it causing the respiratory symptoms?

With regard to the first question, Dr. Macdonald said that as the thymus lay in the anterior part of the mediastinum, at the upper end of the cardio-vascular shadow, and in close apposition to the air-containing lungs which provided such a good radiographic contrast, one would imagine that it could quickly be decided whether enlargement was present; but lateral broadening of the upper end of the mediastinal shadow might be occasioned by other conditions than an enlarged thymus, some of them not even abnormal. Such a broadening was produced in the forced expiration of crying, when the vessels were engorged and the superior *vena cava* was distended. Again, this area appeared broader in the supine than in the prone position, and it was perhaps a little broader in the prone than in the erect posture. Furthermore, in plump children it was broader than in thin children, and was relatively broader the younger the child. More recently, Halsey, using the ciné camera, which gave four to five exposures per second, showed that the gland varied considerably in size with the cardiac cycle. This was a factor at present beyond technical control.

The X ray exposure should be made with the baby in the erect postero-anterior position during full inspiration. It was often exceedingly difficult to expose the film in this phase. It required modern equipment to obtain satisfactory chest radiograms of infants and small children. Moreover, it often required great patience, tact and ability on the part of the technician. This was a feature which might sometimes be overlooked by clinicians.

Referring to the second question, Dr. Macdonald said that if films of the desired technique and quality were achieved, and if there was unquestionably an abnormal broadening of the mediastinal shadow, one had to decide whether this was due to an enlarged thymus. The radiological differential diagnosis included enlarged mediastinal glands (tuberculous or coccal), mediastinal tumour, such as lymphosarcoma, pulmonary consolidation and mediastinal pleuritis. In addition, true asthma, tracheo-bronchitis, congenital atelectasis, congenital heart disease, congenital abnormalities of the larynx and trachea, foreign body in the food or air passages, and diaphragmatic hernia, had all to be considered as likely causes of the respiratory symptoms, and in their differentiation the opinion of a competent radiologist was helpful.

Dr. Macdonald then referred to the third question. He said that if one arrived at the conclusion, from inspection of the postero-anterior film, that an enlarged thymus was present, consideration must be given to whether it, *per se*, was the cause of the symptoms. There was far from

universal agreement on the question whether an enlarged thymus could be the cause of severe or characteristic respiratory symptoms.

A body of opinion had been led to sponsor thymic asthma as a clinical entity by the consideration that in some young children with respiratory symptoms an enlarged thymus could be demonstrated by X rays. After radiation therapy to this radiosensitive structure there frequently occurred both a rapid and permanent improvement in the symptoms and also a diminution in the size of the thymic shadow. Thus it was stated that the causal relationship between the enlarged thymus and the asthma had been established. But it had to be pointed out that this conception had been more vigorously stated a decade or so earlier, when thymic enlargement was diagnosed by X rays much more frequently and erroneously than was the case today.

Dr. Chevalier Jackson, of Philadelphia, believed that there was such a condition as thymic asthma; but he pointed out that lateral enlargement, which was seen in the postero-anterior film, did not cause the symptoms. Dr. Jackson stated that in order that these should occur the thymus had to be enlarged posteriorly and had to encircle the trachea, producing a compression. This tracheal compression Jackson had demonstrated by bronchoscopy. Therefore, if one accepted the view of thymic asthma that received support from Pancoast (also of Philadelphia, and as eminent in radiology as Chevalier Jackson in laryngology), it followed that the radiologist had to attempt to demonstrate the tracheal compression before entering on the diagnosis. The narrowing of the air-containing trachea was better shown in the lateral view, though sometimes it could be seen also in the postero-anterior view. But it was not always easy to show clearly the tracheal lumen in the superior part of the mediastinum. In estimating whether narrowing existed, the clinician had to bear in mind the fact that in full expiration the trachea was normally contracted and bowed, and that the width of the tracheal lumen at the upper orifice of the thorax varied somewhat with extension or flexion of the chin.

Dr. Wasson, of Denver, Colorado, one of the foremost clinical radiologists in the United States of America, and a specialist in radiology of the chest, was a sceptic concerning thymic asthma. Wasson gave treatment other than X ray therapy to a series of patients with so-called thymic asthma. This other treatment was directed towards the removal of broncho-sinusitic infection and tracheal mucus, and it was found that the patients recovered without any X ray therapy. Other X ray therapists had reported that, although there had been no appreciable diminution in the size of the thymus as seen on the film, many of these patients improved clinically after X ray therapy. Others again stated that certain respiratory symptoms had reacted well to X ray therapy over the anterior part of the mediastinum, though the films had failed to reveal any thymic enlargement.

Dr. Macdonald concluded his remarks by pointing out that thymic asthma was under some suspicion as a clinical entity, and it was certainly being diagnosed less frequently than a decade earlier. It had been well proved that true allergic asthma was diminished after X ray therapy to the mediastinum, and Dr. Macdonald felt that many cases of so-called thymic asthma were really cases of allergic asthma, with a concomitant, and perhaps fortuitous, thymic enlargement. He felt, with regard to Dr. McKillop's case, that if an enlarged thymus alone were the cause of the symptoms, the X ray therapy would have been permanently efficacious.

Dr. Ian Wood said that an apparent increase of the size of the shadow to the right side could be explained by slight rotation at the time when the film was taken. He had brought a lantern slide to the meeting, which revealed enormous enlargement of a thymus gland obtained at an autopsy. The infant's thymus came down well over the anterior surface of the heart. It was possible to have great enlargement of the thymus gland without any constriction of the trachea, though its secretion might affect the respiratory system or the breathing might be subject to nervous influence.

Dr. H. Douglas Stephens said that he could corroborate what Dr. Wood had said. He recalled some of the details of a case in which the patient had had a lump rising in the jugular notch with each breath. The child had had practically no thymus gland, but considerable lung fibrosis was present. He also remarked that congenital laryngeal atresia entered the differential diagnosis, but that disorder began at birth. He also referred to an example of mediastinal fibrolipoma, a specimen of which Dr. Reginald Webster had prepared for him.

Dr. J. B. Colquhoun said that at a hospital at which he had been working ten years earlier, on the assumption that the radiologist could diagnose the presence of an enlargement of the thymus gland it had been the routine practice to prepare antero-posterior and lateral skiagrams of the upper part of the chest as a preoperative precaution. This action had been taken because of the desire to avoid sudden deaths, for which the thymus gland had been blamed. Whenever thymic enlargement was reported to be present operation was refused; but unexpected deaths continued to occur. In the end the surgeons did not refuse operation, because they had become convinced that it was not possible to select by radiographic methods those patients who were likely to die under anæsthesia. From that experience Dr. Colquhoun had reached the conclusion that the question of the size of the thymus gland had hitherto been given too much importance.

Dr. Keith Hallam considered that the possible presence of congenital heart anomaly should enter the differential diagnosis.

Dr. Stanley Williams remarked that clinical evidence of irritation of the nerves, as well as of pressure on the trachea, should be expected if enlargement of the thymus was the cause of the baby's illness.

Dr. Colin Ross referred to the autopsy findings in a case of progeria. The patient had died at the age of twelve years; the thymus gland had been found to be eight times the expected weight for age, and to consist of fibrotic material, which made it definitely firm.

Dr. H. Boyd Graham said that he had noted a suggestion in an article that relief of pressure could be effected surgically under local anæsthesia. He thought that that observation might have a practical application if Dr. McKillop's patient became suddenly *in extremis* again. He had also seen references to partial thymectomy, and wondered whether any of the surgeons present had had experiences of surgical methods of treatment.

Dr. Mostyn Powell said that he would have expected evidence of œsophageal obstruction if the thymus was the cause of the trouble. Vomiting, and not tracheal symptoms, was a feature in one case he remembered.

Dr. McKillop, in reply, said that he had formed the impression that the underlying cause of the baby's distress might be one of the neoplasms known to occur in that region. With reference to Dr. Powell's observation, he thought that it might be of interest to record that the attacks were specially apt to occur when the baby was feeding or swallowing. Dr. Ross's positive statement that a certain thymus gland was eight times the expected weight had interested Dr. McKillop, because in spite of some searching he had been unable to discover any authentic figures to indicate the average weight at varying ages. In conclusion, Dr. McKillop stated that he was satisfied that the child had improved after the last exposure to deep X rays. He said that if the members wished it he would report progress at a subsequent meeting.

Tuberculosis in Childhood.

Dr. Reginald Webster reminded the meeting that on the last occasion on which he had appeared before the society he had discussed certain aspects of tuberculosis in childhood. Events since the last meeting had determined that he should introduce the same subject again. It had so happened that autopsy specimens collected during the preceding month, which he had placed on view, had included two examples of extensive tuberculous ulceration of the intestine. One was obtained from a boy aged

eleven years, and the other from a boy aged four years. The extent and character of the ulceration in both were such as to suggest, from *post mortem* appearances, an alimentary and presumably bovine infection.

In the case of the older boy, ulcers of unusual superficial extent, with indications of chronicity in the thickened and indurated edges, affected the lymphoid tissue throughout a long reach of the lower part of the ileum and the whole of the colon. The mesenteric glands were extensively involved in caseating tuberculosis, and, although it could not be said that the tracheo-bronchial glands were free from macroscopically evident tuberculous lesions, these were not obvious. Some search was necessary for their discovery, and they were not comparable in gross characteristics with the tuberculous processes in the mesenteric glands. The fact that Dr. Howard Williams, who made the *post mortem* examination, had been unable to find a primary Ghon lesion in the lungs, seemed further and important evidence that this boy had acquired the tuberculous infection via the alimentary tract.

Dr. Webster said that he had made the *post mortem* examination on the younger boy himself and had found such a massive enlargement of the mesenteric glands as should have been clinically palpable, and of dimensions and character now seen infrequently. The presence of ulceration of the intestine was apparent from the exterior of the bowel, the sites of the ulcers being indicated by depositions of plastic exudate and the occurrence of miliary tubercles in the serous coat of the small intestine. He had determined that tuberculous ulceration affected, at short intervals, the mucous membrane of the small intestine from a point approximately 30 centimetres (12 inches) below the duodeno-jejunal flexure to the ileo-caecal valve. There were also some half-dozen tuberculous ulcers in the colon. He had been unable to find a focus of infection in the lung, other than very sparsely sown small grey tubercles of recent hæmatogenous origin.

In the older boy there was a heavy visceral sowing of miliary tuberculosis, and tuberculous meningitis was present. In the younger boy a very light visceral sowing and no tuberculous meningitis was present.

With respect to the younger of these two boys, the one aged four years, it could be said that the presumption of infection with the bovine type of the tubercle bacillus was likely to be confirmed. In cultures prepared from the mesenteric glands tubercle bacilli had commenced to grow and were making good progress on media containing no glycerine, whereas the cultures on glycerine media were far behind, and it required considerable search to find the organisms in smears made from the surface of such culture tubes. The non-glycerophilic character of the strain appeared to be already established; and that was the important first stage in the identification of the bovine organism.

Dr. Webster went on to say that in the case of the older boy it appeared that experience had once more been fallacious and judgement therefore difficult; for, in spite of the indication of bovine infection provided by the autopsy, the culture of tubercle bacilli which he had recovered from the cerebro-spinal fluid was indubitably of human type. This culture had been obtained from the cerebro-spinal fluid withdrawn by lumbar puncture as a diagnostic measure some ten days before the boy's death, and was therefore ahead of those which he had prepared from the mesenteric glands and lung tissue *post mortem*. The culture of the human type of tubercle bacillus recovered from the cerebro-spinal fluid could be accepted as the infecting organism in this case. In a large series of 1,405 individuals in whom tubercle bacilli had been recovered from more than one source, A. S. Griffiths, the doyen of British workers in this field, did not find one instance of double infection.

Dr. Webster said that no doubt he would be expected to offer some explanation of the apparent discrepancy between the autopsy findings and the bacteriological result as indicating the nature of the infection in this boy. He had frequently seen ulceration of the intestine in undoubtedly human infection in infants and young children who died of disseminated tuberculosis; but such ulcers had always been recent, shallow and limited to the lower part of the ileum, and in distribution and character differed greatly from the specimens he was discussing. He had considered it very probable that in the terminal heavy bacillæmia of miliary tuberculosis an analogy held with typhoid fever, and localization of tubercle bacilli was apt to occur in the spleen and lymphoid tissue of the intestine. In other words, the terminal tuberculous intestinal ulcerations might reasonably be regarded as of hæmatogenous origin.

The question arose as to whether there was any other way in which ulceration in the intestine could be set up by infection with the human type of tubercle bacillus. Dr. Webster said that it was to be remembered that he was discussing tuberculosis in childhood; and the obvious method of intestinal infection in the advanced phthisis of adults, namely, the swallowing of large amounts of sputum laden with tubercle bacilli, did not at first sight appear to come into consideration. Tubercle bacilli had often been detected in tonsils and adenoid vegetations, and in the laboratory at the Children's Hospital he had often demonstrated tuberculous infection of the tonsil by methods which should satisfy the most exacting. From the pharynx the bacilli had easy access to the small intestine, and the gastric hydrochloric acid would offer them no resistance.

The question of the elimination of bacilli and the problem of infectivity in children affected with the type of intrathoracic tuberculosis characteristic of childhood merited close examination. Formerly hilus tuberculosis was regarded as a closed lesion, the elimination of bacilli from which was denied. Now, however, it was conceded that tubercle bacilli might be excreted in all phases of intrathoracic tuberculosis. Dr. Webster had often been impressed with the indications of activity displayed by the small primary Ghon focus, and interpreted central softening and encirclement with peripheral tubercles as evidence that the focus was anything but quiescent. With tubercle bacilli multiplying in the peribronchial lymphatic tissue, and with Boyd's "friendly phagocyte" at hand to transport them within the lumen of the bronchiole, it was not difficult to understand how the organisms might be eliminated from an apparently closed lesion.

At all events, many observations had been recorded in which tubercle bacilli had been demonstrated in the pharyngeal mucus, in the product of lavage of the fasting stomach, and in the fæces of children with the juvenile primary infection type of pulmonary tuberculosis. It was forty years since Meunier, in 1898, had first recommended lavage of the stomach of children during fasting, and a search for tubercle bacilli in the centrifuged deposit of the washings. The method, however, was never employed extensively; but in 1927 Armand-Delille and Vibert reported their results of the examination of 110 children; by Meunier's method they found tubercle bacilli in the products of gastric lavage in 31%. These workers relied on microscopic search only; but with the sensitive methods for the cultivation of tubercle bacilli now available, the method of gastric lavage should become increasingly useful. Three Danish workers, Poulsen, Jensen and Husted, had in 1929 supplemented the microscopic examination of the deposit obtained by centrifuging the gastric washings with cultural methods and guinea-pig inoculation, and had demonstrated tubercle bacilli in the stomachs of eleven out of fifteen children so investigated. The importance of this method of examination lay in the following considerations: (i) it rendered it possible to establish open pulmonary tuberculosis when the diagnosis could not be made by other methods; (ii) it provided evidence that infants with pulmonary tuberculosis were infective, and indicated their isolation; (iii) it was an addition to the means already available for making an incontestable diagnosis of tuberculous pulmonary infection in childhood.

DR. DOUGLAS GALBRAITH said that for a long time he had been impressed by the frequency with which primary foci could be demonstrated in the lungs of children with tuberculosis of bones and joints. He had collected thirty examples at the Frankston Orthopædic Hospital. From these original foci hæmatogenous infection followed; and

he thought it was important to know what would happen to the children in adult life. He hoped to be able to attempt to recover the tubercle bacilli by gastric lavage, and contemplated some such project in the future.

Dr. J. B. Colquhoun congratulated Dr. Webster on the work he was doing on tuberculosis in childhood and on his success in obtaining cultures of the bovine variety of bacillus.

Dr. Webster, in reply, said that he had referred to only one particular type of tuberculosis of the bowel as hæmatogenous, namely, that occurring in babies with widespread miliary tuberculosis.

Pyloric Stenosis.

Dr. J. G. Whitaker said that Dr. Howard Williams would demonstrate a measurement and test of alkalosis and dehydration, which had for a long time been recognized as factors of importance in the problem of pyloric stenosis. Dr. Whitaker drew the attention of members to the originality of the work Dr. Williams had done. He had introduced a scientific and very acceptable means of measuring and combating alkalosis and dehydration, both before and after operation. Dr. Whitaker also desired to draw attention to the excellence of the intravenous therapy which was a strong feature of the treatment of many of the patients at the hospital.

Dr. Howard Williams read a paper entitled "The Control of the Disordered Metabolism in Hypertrophic Pyloric Stenosis of Infancy" (see page 414).

Dr. J. G. Whitaker stated that Dr. Ian Wood had investigated the mortality rate in 100 consecutive cases of pyloric stenosis immediately preceding the series to which he proposed to refer. The babies who formed the subject of Dr. Wood's inquiry had all been submitted to surgery under general anæsthesia, and the mortality rate was 11%. Dr. Whitaker's series consisted of 31 consecutive cases. He had used a high transverse incision and had infiltrated the skin on both sides of the incision, but not in its line, with a local anæsthetic. In the last 20 cases he had had the advantage of cooperation with Dr. Howard Williams in the manner that Dr. Williams had just described. The mortality rate was only 6%, as there had been two deaths in the series. Infection of the wound and difficulty with the preoperative narcosis had been contributory factors.

Dr. Whitaker then discussed a number of points of interest arising out of a review of the detailed results. He said that every operation for pyloric stenosis without the aid of general anæsthesia must be regarded as being in the nature of an adventure. He felt that the mortality rate was likely to be greater if the practice of relying on local anæsthesia extended throughout the country, because there was no doubt that the technical difficulty of the operation was much increased. When the baby was very ill, however, in experienced hands an operation which would be fraught with danger under general anæsthesia could be carried out with satisfactory results under local anæsthesia. The treatment adopted by Dr. Williams was making it much rarer for babies to be presented for operation in a state which could be described as very ill.

The preoperative treatment consisted in: (*a*) dehydration to correct alkalosis (Howard Williams's method), (*b*) suitable feeding, (*c*) elevation of subnormal temperature and attention to fluid requirement, and (*d*) preoperative medication. For a baby weighing 2·7 kilograms (six pounds), 0·4 milligramme ($^1/_{150}$ grain) of heroin had been found necessary, injected fifteen minutes before the operation, to secure a reasonable amount of passivity.

Dr. Whitaker then presented a cinematograph film, in colour, which had been prepared by Dr. M. O. Kent Hughes. It portrayed the steps in the performance of the Rammstedt operation by Dr. Whitaker on the thirty-first baby in his series. Dr. Whitaker drew attention to the introduction of the local anæsthetic around and not in the line of the incision; to the division of the *recti abdominis* muscles between forceps; to the fact that the posterior layer of the sheath of the muscles was very thick; to the free introduction of anæsthetic solution into the peritoneal tissues and the opening in the abdomen. Other features were the presentation of the liver, which filled the field in the peritoneal window, and the manner in which the pyloric tumour could be brought into view and rested against the liver. The tumour was divided along its long axis with the edge of a scalpel and was opened with the special forceps designed by Dr. Mostyn Powell, the division being completed at the duodenal end by a pair of fine blunt-nosed Spencer Wells forceps. Dr. Whitaker also remarked on the ease with which the wound could be closed. He stated that as long as two years after the operation by means of the transverse incision the healed scars had remained quite sound and had not shown any tendency towards the formation of a hernia at the operation site.

Dr. A. P. Derham said that he had not realized that Dr. W. G. D. Upjohn had observed that hypertrophic pyloric stenosis was a spring disease; but it was noticeable in the wards during the few weeks prior to the meeting that there had been quite a number of patients with that condition. With reference to Dr. Howard Williams's work, Dr. Derham said that the method not only made the operation safe, but had in some instances made it possible. Early in September Dr. Derham had seen a baby, four days old, in an extremely serious condition due to hæmatemesis and melena. The child had survived and improved considerably after treatment by Dr. Stanley Williams, who had given an intravenous transfusion of blood from the father. After an interval of eight or nine days the baby had again become very ill from vomiting due to hypertrophic pyloric stenosis. After Dr. Williams had given saline solution with added glucose for thirty-six hours by the continuous intravenous drip method, the baby had been subjected to operation. This was performed by Dr. Whitaker and had been followed by uneventful recovery.

Dr. Ian Wood said that it was of interest to consider the evolution of the treatment of pyloric stenosis. He had learned not to waste time, on account of increased operative risk; and at that time the babies were liable to be rushed onto any operating table. When he had gone to the Great Ormond Street Hospital he had been politely thwarted from his policy of haste and had had to wait two or three days. During this time fluid was introduced subcutaneously and frequent feedings were administered. Since his return to Melbourne the London practice had been endorsed and justified, and had culminated in the present work of Dr. Howard Williams. It was doubtful whether the dehydration and alkalosis could be satisfactorily managed by the introduction of fluid subcutaneously and orally only. The addition of glucose amounting to 5% of the fluid introduced intravenously was advisable. Perhaps Dr. Howard Williams had omitted reference to the glucose for the sake of clarity of description of his method.

Dr. Stanley Williams referred to the tremendous amount of work involved in what was behind the simple description of Dr. Howard Williams's new method, which was of world-wide importance. He wondered whether the continuous drip administration of fluid commenced before operation was continued during the operation and for a short time afterwards. Dr. Williams congratulated Dr. Whitaker on the low operative mortality rate for thirty-one consecutive cases. He thought that that information was also of great importance.

Dr. Colin Ross also congratulated Dr. Whitaker and Dr. Howard Williams. He said that while he was at Birmingham between sixty and ninety babies annually had caused him worry over pyloric stenosis. During the period covered by his experience he had reviewed the patients in three groups. The earliest group had consisted of babies operated on under general anæsthesia without supportive treatment; and the mortality rate was 29%. The average condition of the babies when they arrived at the hospital was incredibly worse than that of the babies seen at the Children's Hospital, Melbourne. The second group comprised those more generally operated on under local anæsthesia, but largely without supportive

treatment; the mortality rate had fallen to 18%. In the last and more recent group dehydration and the use of glucose had been the rule; but there had been a reversion to general anæsthesia. Of the patients 60% had been operated on under general anæsthesia and 40% under local anæsthesia; the mortality rate had fallen to the satisfactory level of 9%.

Dr. Ross made one suggestion, which concerned the occurrence of œdema during the continuous intravenous introduction of fluid. The question of the blood protein content had to be watched. Irrespective of other biochemical imbalance, protein depletion might be present; this could be countered by the transfusion of whole blood or blood serum in addition to the other fluids.

DR. DOUGLAS GALBRAITH said that he had followed with interest through the years the gradual departure from the purely mechanical conception of pyloric stenosis to the recognition of the metabolic defect and study of the chloride metabolism. In 1933 Morris and Graham had published work on the measurement of alkalosis and acidosis, in which the chloride metabolism had been studied. Variations might occur in the chloride content of the blood or urine, and there might be increased retention or depletion of chlorides. Though vomiting was the apparent cause of the loss of chlorides, the two things did not always run parallel; the chloride loss did not occur, for instance, in tuberculous meningitis. Dr. Leonard Findlay had reported a case in which there was no vomiting, but the chloride loss was established all the same; there was a depression of the respiratory rate, and an estimation of the alkali reserve was in favour of chloride loss. In addition, a typical tumour was palpable and classical visible peristalsis was present. The baby had died and definite obstruction had been found. If the disordered chloride metabolism was not due to vomiting, it was of interest to consider other possible causes. It was known that the injection of histamine stimulated secretion of hydrochloric acid and thus caused chloride depletion. Dr. Galbraith suggested the possibility that at the operation the surgeon might not only relieve the mechanical obstruction, but might cut through a nervous plexus or in some manner produce a biochemical change. At all events, in the management of pyloric stenosis it was ideal to combine the work of surgeon, physician and biochemist.

DR. ROBERT SOUTHBY commented on the exclusion from classical descriptions of pyloric stenosis of a symptom many of the mothers mentioned to the doctor. He referred to the diminution or lack of urinary output; the baby might not have passed urine for twenty-four hours. Unexplained occurrences, such as hyperpyrexia and convulsions of an unexpected nature, sometimes causing death, might be due to biochemical changes. Premedication by means of brandy and glycerine had often been practised in the past, and might be helpful in rendering operation under local anæsthesia more satisfactory.

DR. H. DOUGLAS STEPHENS said that he had found the debate very interesting and had appreciated the excellence of Dr. Howard Williams's work. The advantages of local as against general anæsthesia and of the transverse as against the vertical incision were discussed by Dr. Stephens at some length. While thoroughly appreciative of the work done, he was not sure of the absolute value of restoration of the alkali reserve before operation. He had in his time operated on some desperately ill babies and had obtained satisfactory results by giving a little saline and glucose solution rectally and some fluid subcutaneously. Alteration of the alkali reserve, while doubtless of benefit, might not be essential. He had performed many of the operations under local anæsthesia; but he preferred general anæsthesia for babies weighing more than 2·7 kilograms (six pounds). Although he had used premedication with brandy, glucose and sugar, as well as with these substances in combination with "Nembutal" and other substances, he had been unable to standardize preoperative medication to his satisfaction. In fact, what he had done had at times seemed to contribute towards the deaths that had occurred. Many of the patients had done well; but sometimes struggling had occurred, and there was a tendency for the wounds to break down after infiltration with the local anæsthetic. There was a danger of sepsis that might reach the peritoneum, and struggling resulted in pushing out of the omentum, which prolonged the operation. If the baby weighed 2·7 kilograms (six pounds) or over, it was likely to stand anæsthetization with ether quite satisfactorily. Dr. Stephens preferred an incision in the peritoneum, below the falciform ligament, through a paramedian incision in the skin and subcutaneous tissues. He liked to perform the operation left-handed; when he pulled the stomach out the pylorus followed and could be readily incised with the left hand. He was not inclined to alter the vertical incision, which had proved quite adequate for many years. He wondered whether the occurrence of hyperpyrexia after operation was related to the excessive application of heat to the patient's body.

Dr. Williams, in reply, expressed his appreciation of the way in which his contribution had been received, and referred to the excellence of the work of Miss Green, the biochemist at the hospital, who had cooperated with him most ably. He observed that hyperpyrexia had not occurred in any of the twenty cases that had formed the basis of his study. Attacks of tetany had been encountered three times in the year prior to the introduction of the continuous drip therapy, and were indicative of alkalosis. One was fatal; but the other patients had been relieved by the administration of carbon dioxide. They had left the glucose out of the solutions used in order to study the effect of sodium chloride without any such complicating factor.

DR. J. G. WHITAKER said that the babies still received their brandy. It was administered with sugar on a comforter placed in the mouth during and after the operation.

Obituary.

WALTER HENRY TOFFT.

WE are indebted to Sir John Ramsay for the following account of the career of the late Dr. Walter Henry Tofft.

With the passing of Walter Henry Tofft on January 29, at the age of seventy-six years, the grand old school of country practitioners has been depleted. Nearly half a century of his life of active practice was spent at Campbell Town, Tasmania—the centre of the breeding of high-class merino sheep. The descendants of the pioneers of more than a century ago were his patients, as also the working people of the town and district. In his professional work he knew no class distinction.

The son of Major Tofft, an officer of the British Army in India, he was born in that country, and, though he left it at an early age, was able to converse in Hindustani throughout his life.

Migrating to Tasmania, he was educated at the High School, Hobart, obtained his A.A. degree and won the Tasmanian Scholarship, which helped him to pursue his student career at the University of Edinburgh. After graduating M.B., C.M. in 1887, he was house physician at the Edinburgh Royal Infirmary to Professor Fraser, and subsequently resident physician at the Glasgow Royal Infirmary for a time. After short experiences of private practice in Corowa, New South Wales, and Newtown, Tasmania, Dr. Tofft ultimately settled in Campbell Town, Tasmania. His district extended for more than thirty miles in several directions. His ready response to calls, however distant, and whether a fee was forthcoming or not, endeared him in the hearts of his people. Travel was not easy in those days, but whether for miles he walked or drove or rode his bicycle, the dictates of duty were uppermost in his mind. The story is told of one of his journeys on his bicycle, in which he caught up with a number of competitors in a cycle road race and "headed the field".

He realized the possible usefulness of the elevated midlands of Tasmania in the treatment of tuberculosis, and built a sanatorium of individual huts. Many of the past inmates even to this day have cause to thank him for their restored health.

The clarion call of the Great War to the son of an army officer brought him into the Australian Imperial Force, in which he served till the end of the War, attaining the rank of major and receiving the decoration of M.B.E. from His Majesty the King. He served first at Woodcote Park Convalescent Hospital and then successively at Wareham, Codford and Hurdcott Command Depots, of the last named of which he was senior medical officer. His wife and unmarried daughter followed him to England, where both gave useful service. His wife, a trained nurse, worked with the Red Cross, and his daughter occupied a position at the War Office. On his return he added sheep-growing to his many other occupations, and "Brookdale", the site of his sanatorium, became the centre of his medical and pastoral activities.

In spite of his busy practice he found time to interest himself in local public affairs. He was warden of the municipality for twelve years, and a member of the Senate of the University of Tasmania. He had been medical officer to the local hospital for twenty-four years prior to the War, and health officer to the district for a longer period. He was a crack rifle shot and often represented Tasmania in the interstate competitions. He was the ideal country practitioner, beloved and respected by all who knew him. When "The Last Post" was sounded at the graveside the great crowd that came from near and far was sufficient evidence of the high esteem in which he was held. Our sympathy is extended to his widow and daughters, who helped him in his life's work.

Correspondence.

THE TREATMENT OF ELECTRIC SHOCK.

Sir: The Council of the Queensland Branch of the British Medical Association has received a reply from the British Medical Association, London, giving the opinion of an expert consulted by the Medical Research Council, as follows:

> If the pupil is widely dilated and keeps so, recovery will not take place in ordinary cases, but in electric shock this excellent test may not be trustworthy, as the centre for the pupil may be out of action owing to the shock, just as the respiratory centre may be. I therefore, on reconsideration, think that the rule of going on with artificial respiration till *rigor mortis* sets in is simple and acceptable, although it will cause much waste of energy, as recovery after a long period is very rare. For long-continued artificial respiration, Eve's rocking method may be used (as an alternative to the Schafer method); it saves the subject from injury and the operator from fatigue, and is efficient both in aeration of the lungs and in furthering the circulation by the rhythmic pull and push on the diaphragm.

The opinion quoted above will probably be of interest to your readers.

Yours, etc.,

Horace W. Johnson,
Honorary Secretary.

Brisbane, B.17,
February 27, 1939.

THE TREATMENT OF INFANTILE PARALYSIS.

Sir: Rest, support and movement are the three essentials. Unfortunately the two former have been unduly emphasized during recent years in Melbourne. When I joined the staff of the Children's Hospital in 1893, acute cases were few and far between. Rest was limited to about two weeks; support and movement were not used. By the time the epidemic of 1908 arrived, rest was continued for several months; support and movement, both active and passive, were in general use. Mackenzie (1910) promulgated his absurd theory that infantile paralysis was purely a muscle affection, and stated that the then and now accepted pathology was wrong. He insisted on prolonged rest for years, which was further elaborated in 1925 by Dr. Macnamara, who eventually kept patients closely confined for periods up to five or seven years without any movement at all.

Sister Kenny has certainly helped to break down this pernicious form of treatment, though the pendulum swung too far. However, in my opinion, too much reliance is still placed on too long periods of rest. Support by extensive splinting is also overdone. The profession has forgotten the precepts of T. S. Ellis, that the best neutral position for the muscles acting on a joint is midway between its extremes of movement. For instance, the muscles of a foot at right angles are not at rest, but they are so in slight plantar flexion, the position the foot assumes when it is swung forward.

The positions of zero enunciated by Batten and adopted by W. C. Mackenzie are quite wrong. Whether we are dealing with a shoulder, knee or foot, though only one muscle may be severely affected, it is a good rule to realize that all the muscles are or may be affected. Beyond moving joints passively to prevent contraction, I do not think much good is done by so-called reeducation of muscles for the first few months.

Pain and spasm are better and more quickly relieved by diathermy in the acute stage. You cannot apply diathermy too early to both the cervical and lumbar enlargements. The quicker you get rid of the œdema in the spinal cord, the better. Rest helps, but diathermy dispels.

Yours, etc.,

W. Kent Hughes.

Melbourne,
February 27, 1939.

Sir: Following on the report of Doctors Forster and Price on an investigation of cases of poliomyelitis in which the "Kenny system" of treatment was used, may I ask, in view of the rather extensive antagonism to this method of treatment in early stages of our recent poliomyelitis epidemic, that all readers interested in poliomyelitis should pay particular attention to the conclusion of the report. One very important fact has been omitted from the report. It is this: The treatment of these cases referred to was carried out by trained nurses selected in Melbourne by Sister Kenny, and not by trained physiotherapists. One nurse had previous experience in Brisbane under Sister Kenny's supervision and was made responsible for the supervision and training of the nurses at Hampton. The trained nurses were not selected for their previous knowledge of orthopædic nursing or experience in poliomyelitis. Those familiar with any method of muscular reeducation will at once admit that muscular reeducation is an art as well as a science, and for those raw recruits to have obtained results as published commands at once our attention and admiration.

Yours, etc.,

Rae W. Dungan.

19, Selbourne Road,
Toorak,
Victoria.
February 27, 1939.

ACUTE HÆMOCYTOLYTIC ANÆMIA.

Sir: In your recent number of the 25th instant I read with interest the description of a case of "Acute Hæmocytolytic Anæmia", by Dr. D. G. Hamilton.

I am in agreement with all his conclusions, except that of the deranged kidney function. It is known that the healthy kidney will excrete hæmoglobin, but the acid nature of normal urine causes the formation of insoluble acid hæmatin, excess of which in the renal tubules can cause anuria, or at least fairly considerable renal damage.

Such a state of affairs was existing at the time of the patient's admission to hospital; in fact in the history a point is made of it. There is no mention of the alkalinization of the urine or the giving to the patient of excess fluids (compare blackwater fever). Apparently all attention was centred on the catastrophe which had befallen the erythron—the side-effects would look after themselves.

Immediate transfusion began the arrest of the hæmocytolysis, so vividly described, but one is forced to remark that it was rather fortunate for the patient that the kidney function recovered as remarkably as it did. It would have been a pity if the child had died of uræmia when such a lot of trouble had been taken.

Yours, etc.,

C. Leleu.

R.A.A.F. Station,
Point Cook,
Victoria.
February 27, 1939.

The Royal Australasian College of Physicians.

ANNUAL MEETING, MELBOURNE, 1939.

The first annual meeting of the Royal Australasian College of Physicians will be held at Melbourne on Thursday, Friday and Saturday, March 23, 24 and 25, 1939. The programme will be as follows:

Thursday, March 23.

2 p.m.—First scientific session, to be held in the hall of the Royal Australasian College of Surgeons.

2 p.m.—"Significance of Myocardial Scars", Dr. T. E. Lowe.

2.30 p.m.—"Recent Advances in the Treatment of Insanity". Two papers will be read, as follows: 2.30 p.m., "Treatment with 'Cardiazol' and 'Somnifane' ", Dr. H. F. Maudsley; 3 p.m., "Treatment with Insulin", Dr. John K. Adey. The discussion will be opened by Dr. J. F. Williams.

4 p.m.—Afternoon tea.

4.15 p.m.—"Some Aspects of Tuberculosis", Dr. Reginald Webster.

4.45 p.m.—"Radiation Treatment of Thrombocytopenia", Professor J. V. Duhig.

5.30 p.m.—Late afternoon party at the Windsor Hotel, to which all Fellows and Members of the College and their wives are invited by the Victorian State Committee.

Friday, March 24.

10 a.m.—Council meeting, to be held in the library of the Royal Australasian College of Surgeons; the admission of members will take place at this meeting.

12.45 p.m.—Luncheon for Fellows and members at the Oriental Hotel.

2 p.m.—General meeting, to be held in the hall of the Royal Australasian College of Surgeons.

3 p.m.—Second scientific session, to be held in the hall of the Royal Australasian College of Surgeons.

3 p.m.—"Problems of Carbohydrate Metabolism, with Special Reference to Absorption from the Alimentary Canal", Dr. Colin Ross.

3.30 p.m.—"The Tuberculin Patch Test", Dr. D. J. Anderson.

3.45 p.m.—Afternoon tea.

4.15 p.m.—"Regional Entero-Colitis", Dr. Julian Smith, junior.

4.45 p.m.—"Cystic Disease of the Lung", Dr. W. J. Newing.

8.15 p.m.—Annual meeting, to be held in the Wilson Hall of the University of Melbourne. His Excellency the Governor of Victoria and Lady Huntingfield will be present at this meeting, and the Commonwealth Government and Victorian State Government will be represented. A public address, entitled "The Physician, his Life and Times", will be given by Dr. S. A. Smith.

Saturday, March 25.

9.30 a.m.—Third scientific session, to be held in the Lecture Hall of the Walter and Eliza Hall Institute of Research in Pathology and Medicine.

9.30 a.m.—Presentation of clinical cases by (i) Dr. Hume Turnbull, (ii) Dr. John Hayden, and (iii) Dr. S. O. Cowen.

10.30 a.m.—Demonstrations by Dr. Charles Kellaway, Dr. F. M. Burnet and members of the staff of the Walter and Eliza Hall Institute.

A brief cinematographic demonstration of a case of athetosis will also be given by Dr. Eric Susman during one of the scientific sessions.

Throughout the meeting pathological demonstrations of current original work in Melbourne or exemplifying the subjects discussed at the scientific sessions will be set out in the library of the Royal Australasian College of Surgeons. These exhibitions can be viewed before or after the meetings, or on Friday morning. A trade demonstration will also be arranged for March 23 and 24 on the upper floor of the Royal Australasian College of Surgeons.

Corrigendum.

Dr. S. V. Marshall has drawn our attention to an error occurring in his article on "Pentothal Sodium" anæsthesia, appearing in the issue of March 11, 1939. At page 385, line six, the words "Children about ten years of age" should read "Children under ten years of age".

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

McGrath, Walter Stafford, M.B., B.S., 1930 (Univ. Sydney), 147, Anzac Parade, Kensington.

Campbell, Neil Douglas, M.B., B.S., 1939 (Univ. Sydney), St. George District Hospital, Kogarah.

Finckh, Dorrie Alfreda, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

Davis, Eric Lewis, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

O'Reilly, Margaret Linnell, M.B., B.S. (Univ. Sydney), Queen Victoria Hospital, Mint Place, Melbourne, C.1.

The undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Fotheringham, James David, M.B., B.S., 1937 (Univ. Adelaide), Henley Beach Road, Torrensville.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Campbell, Allan Gordon, M.B., B.S., 1938 (Univ. Adelaide), 108, Finniss Street, North Adelaide.

Tamblyn, Eric Joseph, M.B., B.S., 1936 (Univ. Adelaide), Uraidla.

Hamp, Edward James Chipp, M.R.C.S., L.R.C.P. (London), 1929, 28, East Terrace, Kensington Gardens.

Irwin, William Morris, M.B., B.S., 1938 (Univ. Adelaide), Adelaide Hospital, Adelaide.

Diary for the Month.

Mar. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Mar. 22.—Victorian Branch, B.M.A.: Council.
Mar. 24.—Queensland Branch, B.M.A.: Council.
Mar. 28.—New South Wales Branch, B.M.A.: Council (Quarterly).
Mar. 30.—South Australian Branch, B.M.A.: Branch.
Mar. 30.—New South Wales Branch, B.M.A.: Annual Meeting.
Apr. 4.—New South Wales Branch, B.M.A.: Council.
Apr. 5.—Western Australian Branch, B.M.A.: Council.
Apr. 5.—Victorian Branch, B.M.A.: Branch.
Apr. 6.—South Australian Branch, B.M.A.: Council.
Apr. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Apr. 14.—Queensland Branch, B.M.A.: Council.
Apr. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee and Ethics Committee.
Apr. 19.—Western Australian Branch, B.M.A.: Branch.
Apr. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 26.—Victorian Branch, B.M.A.: Council.
Apr. 27.—New South Wales Branch, B.M.A.: Branch.
Apr. 27.—South Australian Branch, B.M.A.: Branch.
Apr. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. E. C. M. Wallace has been appointed a Medical Officer in the Medical Branch of the Department of Public Instruction of New South Wales.

* * *

Dr. W. B. C. Gray has been appointed Medical Officer of Health to the Youanmi Local Board of Health, in accordance with the provisions of *The Health Act*, 1911 to 1937, of Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xxi.

Australian Army Medical Corps: Medical Officer.

Aramac Hospital, Aramac, Queensland: Medical Officer.

Austin Hospital for Cancer and Chronic Diseases, Heidelberg, Victoria: Honorary Officers.

Royal Society of Medicine, London, England: William Gibson Research Scholarship for Medical Women.

Saint Vincent's Hospital, Sydney, New South Wales: Honorary Officers.

The Eastern Suburbs Hospital, Waverley, New South Wales: Honorary Assistant Surgeon.

The Rachel Forster Hospital for Women and Children, Sydney, New South Wales: Medical Officers.

Medical Appointments: Important Notice.

Medical Practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Orginal articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.